The
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VOLUME 25 NUMBER 11-12 NOVEMBER, 1953

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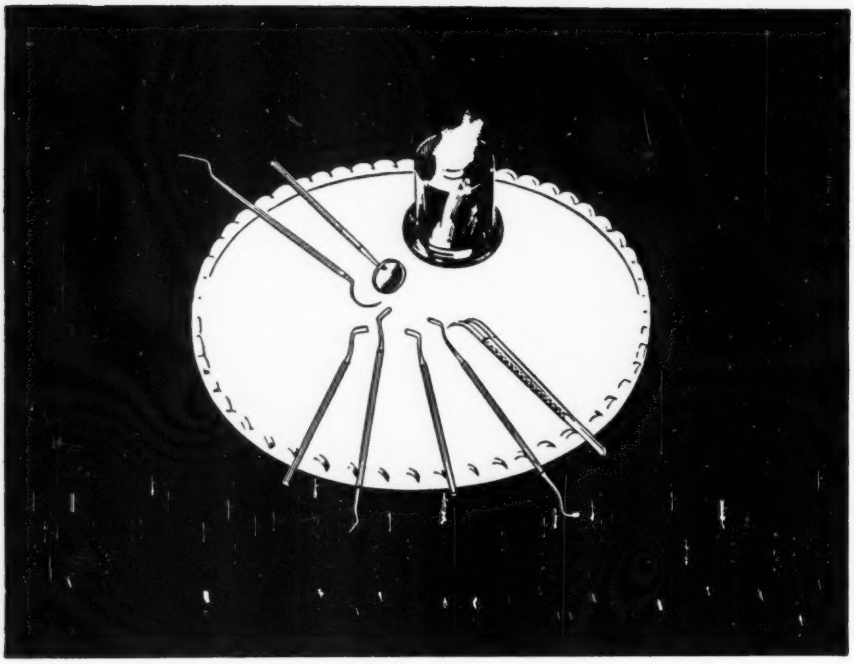

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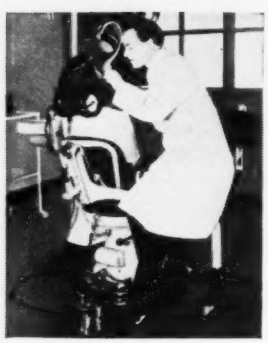

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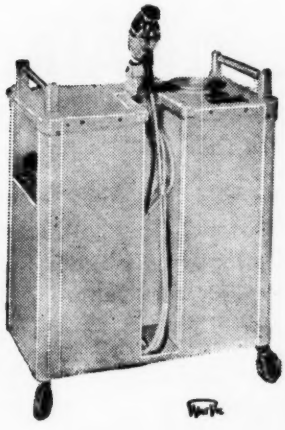

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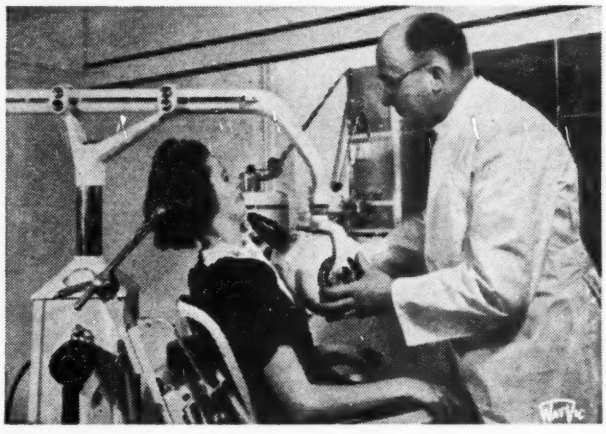

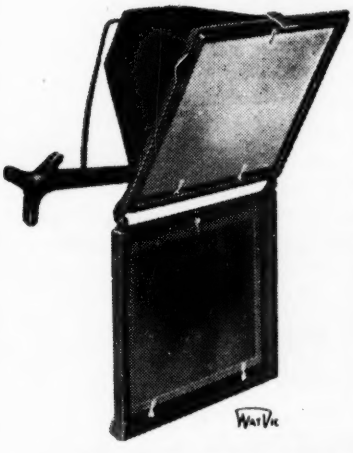

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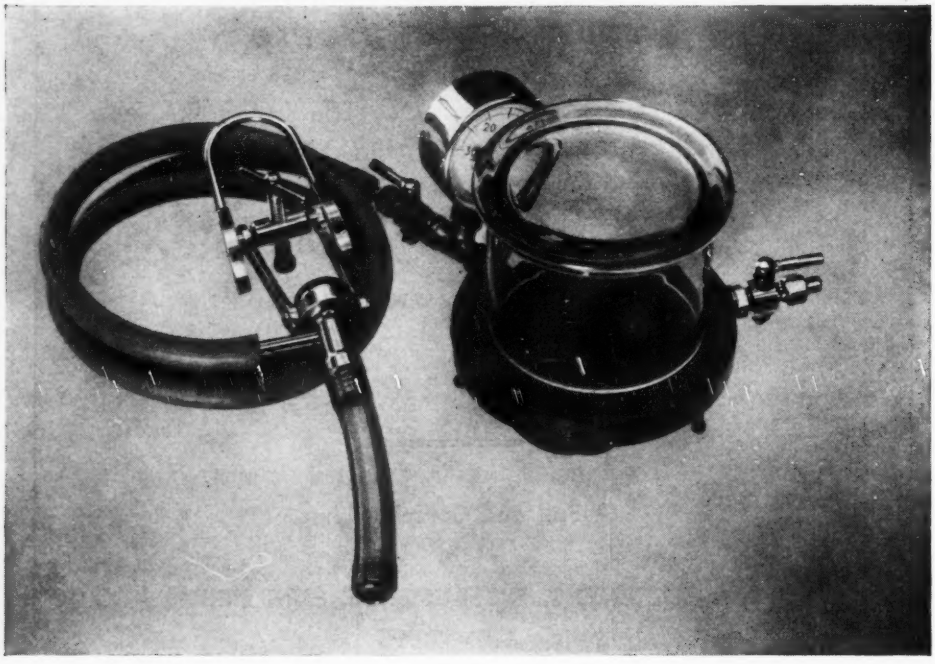

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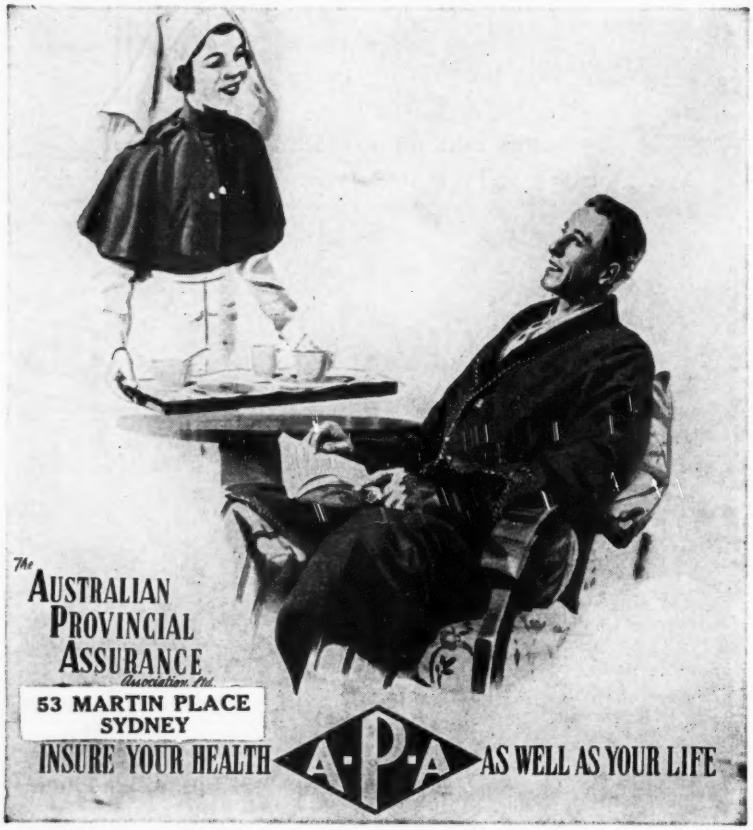

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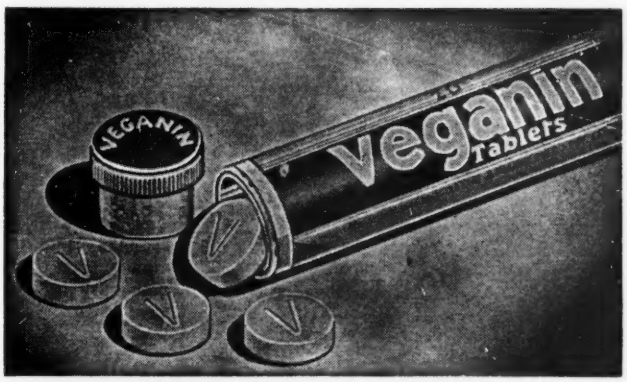

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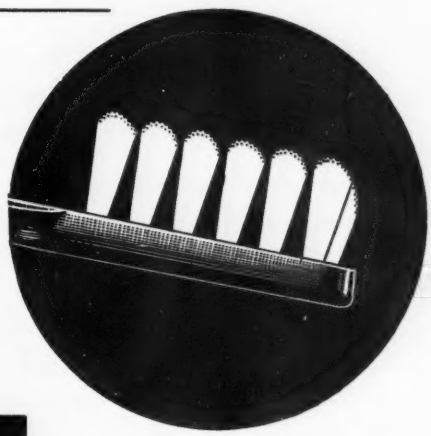

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Periodontal Disease— A Practical Approach to its Treatment and Prevention*

PART I.

RECOGNITION AND TREATMENT OF EARLY PERIODONTAL LESIONS OF LOCAL ORIGIN WITH SPECIAL REFERENCE TO ORAL HYGIENE.

Sydney Levine, M.D.S. (Syd.)

So many mouths show evidence of untreated early periodontal lesions that one is forced to the conclusion that there must be many who fail to recognise these early pathological changes in the gingival tissues and tooth supporting structures; or, if they do recognise them, then they do not appreciate that if left untreated these lesions inevitably lead to a periodontitis with its appalling effect on the dental arch.

This paper will discuss briefly these early lesions which to some may appear too insignificant to worry about but which in reality are the beginnings of much more serious disturbances, and also to draw attention to certain other lesions which may seem too advanced for conservative treatment, but which in fact are not. The writer wishes especially to stress the part that oral hygiene can play in eradicating these disturbances.

Oral hygiene means much more than merely brushing the teeth. It consists essentially of two parts:—

1. That brought about by the dentist in three ways: (a) by restoring where possible all carious teeth to normal by the use of correct operative procedures, and replacing missing teeth by correctly designed prosthetic appliances; (b) by correcting all faulty dental work that may have been performed previously, such as correction of contact points and eradication of overhanging margins, etc.; and (c) by removing all calcified deposits and other debris from the teeth.

2. That brought about by the patient himself, who must be taught how to keep the teeth free from debris and deposits and how to apply stimulation to the gums to maintain a keratinised layer of tissue on them.

This keratinised layer of epithelium, which Gottlieb and Orban[1] say should extend to the bottom of the gingival crevice as well as covering the gingival tissue generally, plays a very important part in periodontal health. Kronfeld[2] stresses this when he says that

> . . . hornification of the gingival mucosa is an important factor in the maintenance of healthy normal gingivae; in its absence, the gingivae are abnormal, red and swollen and bleed easily at the slightest mechanical injury.

Fish[3] states also that stratified squamous epithelium if subject to friction becomes keratinised and that the gingival tissue is no exception, and he goes on to say, "The patient has only to be told how to apply friction, when, where and with what" to keep the tissues keratinised.

The lesions to be discussed presently will generally respond to attention to oral hygiene, as defined above.

Firstly, let us look at the normal periodontium. Obviously, one must be perfectly familiar with the normal to recognise at once those variations from it which, if left untreated, lead to serious periodontal disturbances.

Fig. 1.—Normal interproximal bone in the posterior region.

Normal gingival tissue is generally pink, firm and stippled and hugs the necks of the teeth. The crevice can be explored with a blunt probe without discomfort or bleeding. The ideal crevice depth is zero, but there may be no abnormality with greater depths if the gingival tone is good and the underlying bone normal. In dark skinned people, however, the gums will be much darker in colour and still normal, so it is important to establish a normal gingival colour for each patient. Normal interproximal bone in its x-ray appearance has a clear cut upper edge, generally with a distinct layer of compact bone (Fig. 1). In the lower anterior region, especially in young people, the bone may come to a point. Normal periodontal membrane has an even thickness throughout its length with a well defined lamina dura.

The following is a short review of some of the lesions of the type previously mentioned, listed under causes.

* Lecture in three parts delivered to the Australian Dental Association, New South Wales Branch, on 25th August, 1953.

Periodontal lesions associated with poorly executed fillings, inlays, crowns and carious teeth.

These factors operate in many ways: The restoration may be too large and overflow the cavity or be depressed below the cavity surface. It may make a faulty contact point with a neighbouring tooth or with another filling, or it may overhang in the region of the gingival margin. All these things help to retain food and debris about the gingival margin and cause food impaction in the interproximal space. The overhanging margin in the interproximal space may irritate and ulcerate the gingival margin during mastication and brushing. The restorations may lack or have improperly shaped marginal ridges and this may cause interproximal food impaction.

Fig. 2.—Overhanging margin and early bone loss.

Carious teeth constitute a threefold factor in gingival irritation. In the first place, they cause food retention and then impaction interproximally; secondly, the jagged rough edges of the carious cavity cause irritation of the gingival margin during mastication and brushing, and thirdly, if the cavity is deep and painful the patient does not chew in this area, with subsequent inflammatory disturbance from disuse.

In the above cases the gingival margin will be swollen, detached from the tooth surface, purplish in colour or even bright red. In any case there will be a departure from the normal. If the condition has existed for some time, the x-ray will show loss of bone horizontally; the compact edge will have disappeared and its upper margin will appear moth eaten (Fig. 2). The cause of this condition should not be difficult to find. It must be corrected at once to prevent further food impaction and bone loss. The patient then should be instructed in home care. This is indeed an early lesion which can be promptly treated. The writer emphasises that food impaction creates a vicious circle in relation to damage to the periodontium; firstly it produces an inflammatory reaction which causes loss of the alveolar crest and enlargement of the papilla which in turn produces more food impaction and so it goes on.

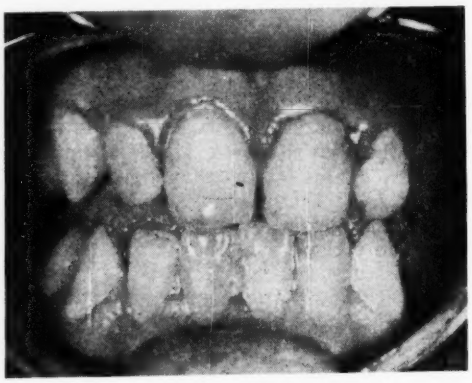

Fig. 3.—Mater a alba.

Lesions associated with poorly designed partial dentures and bridgework.

A denture may lack occlusal rests and be so constructed that it fits into every gingival crevice and because of the pressure it exerts a marginal gingivitis is set up. It may also have ill fitting clasps and occlusal rests or faulty contact points with the remaining natural teeth and so cause food retention with subsequent food impaction. Then again the clasps may impinge on the base of the gingival crevice and so cause an irritation. In these cases the treatment is obvious.

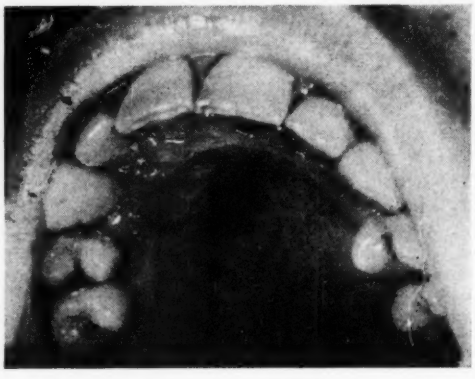

Fig. 4.—Patient has neglected one side.

Lesions associated with subgingival calculus and lack of oral hygiene generally.

When the patient does not attend to his own oral hygiene, there may be no calculus, but

the gingival tissues will have a neglected discoloured look with oedema of the interdental papillae and there will be present soft debris and materia alba (Fig. 3). The gum margins will be detached from the necks of the teeth and will bleed easily. This departure from the normal should be noted, and proper attention to the patient's home care will correct this. Often this is seen on one side of the mouth only; right handed people may neglect the right hand side and left handed the left (Fig. 4).

Fig. 5.—Subgingival calculus between first and second molars and early bone loss.

The lesions associated with subgingival calculus may take two distinct forms. The first type should be recognised at once from the x-ray picture. The bone loss will be similar to that previously described and the calculus will show as spicules of light grey material adhering to the tooth surface in the interproximal space below the contact point (Fig. 5). The gum may be engorged and purplish-

Fig. 6.—Subgingival calculus and lack of oral hygiene generally.

red in colour and have an unbrushed neglected appearance (Figs. 6-8). The treatment is a thorough prophylaxis removing all the calculus, instruction in proper use of the toothbrush and possibly interdental stimulation. If the papilla (especially in the molar area) is detached from the tooth to such a degree that there are separate labial and lingual papillae then these papillae may have to be exercised to get the best effect from stimulation.

The second type is often seen in the anterior region in both the upper and lower jaws. In these cases the lesion is of a hyperplastic nature. There will often be an increase in the size of the gingival tissue to such an extent that bulbous masses may partly cover the crowns, and some of the enlarged tissue may be ulcerated. Bleeding may readily occur with soreness at the sites of the ulcers. Sometimes this hyperplastic tissue has a normal pink colour and there is quite often no bone loss (Figs. 9 and 10).

So many of these latter types of cases, although they seem too advanced for conservative treatment, will respond readily to a thorough prophylaxis and correct toothbrush drill and use of interdental stimulation. Of course, if this were to fail, gingivectomy could be performed and then home care instituted to maintain the tissue in a normal healthy state.

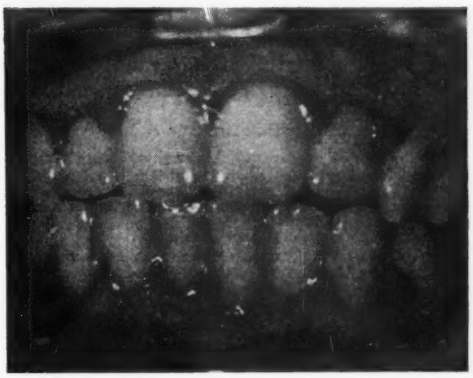

Fig. 7.—Subgingival calculus and lack of oral hygiene generally. $\overline{1}$ shows evidence of traumatic occlusion.

Lesions associated with the wearing of orthodontic appliances.

These are brought about in two ways: firstly, the appliance makes the patient's oral hygiene difficult and, secondly, it may be ill fitting and so be an irritant. Special care is needed in these cases and when the appliance is removed even for short periods prompt attention should be given to oral hygiene.

Lesions associated with overcrowded teeth, malplaced teeth and irregularly shaped teeth.

These conditions cause retention of food and make cleaning difficult. The gums may appear reddish or purplish, possibly engorged and bleed easily. In well cared for mouths the

condition will be seen in the areas of such teeth only. In any case there will be a departure from the normal. Treatment is careful prophylaxis because there may be subgingival calculus and special attention to brushing in these areas and, if possible, orthodontic treatment (Fig. 9).

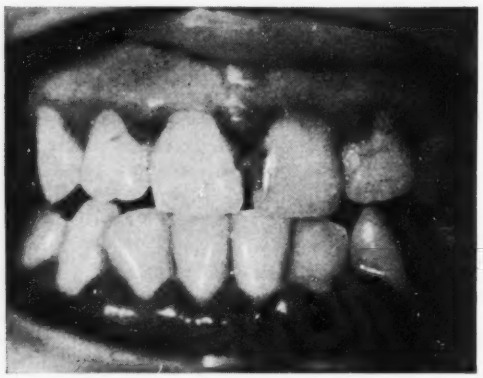

Fig. 8.—Subgingival calculus and lack of oral hygiene generally.

Lesions associated with mouth breathing and marked disproportion between the jaws as in Class II and Class III malocclusions (Fig. 11).

The enlarged gingivae so frequently associated with mouth breathing can be reduced considerably and kept in check by attention to oral hygiene.

In this regard Fish[4] says

> The chronic hypertrophic marginal gingivitis so often associated with this condition (mouth breathing) is no doubt in part due to the exposure of the gum margins to desiccation dust and infection generally, but in view of the fact that it can be cured by the same means as other types of gingivitis it must be attributed to the lack of lip tongue and food friction which in turn is the result of the disproportionate growth of the jaws so often associated with this condition.

Fig. 9.—Hyperplastic reaction with subgingival calculus. Note irregularity of the lower teeth.

Lundquist[5] believes that the important factor in the neglected chronic marginal gingivitis is that the lymphatics are blocked and that it is only through the lymphatics that the extravasated cells and fluids can be returned to the blood vessels. He says that this readily explains the swollen gingivae seen in dirty mouths and would later develop into a fibrosis. He further states that massage and motion are the best means of promoting lymph drainage and so advocates this as a primary factor in treatment. In children, he believes that there is little more than detachment of the soft tissues from the crowns and overgrowth of the tissue (that is, oedema with blocked lymphatics) which later becomes fibrous. In the adult there is a deeper crevice

Fig. 10.—Hyperplastic reaction with subgingival calculus.

with greater inflammation but less oedema. So the writer believes that in these conditions, after a thorough prophylaxis, massage should be tried first. If this fails because of too

Fig. 11.—Gingivitis associated with mouth breathing and malocclusion.

great a degree of fibrosis, then surgery can be performed and then special attention given to home care to prevent a recurrence.

It is in these Class II and III cases mentioned above that orthodontia plays such an important role.

Gingivitis due to accidental trauma.

This may be caused by forcing under the gum margin such things as a toothbrush bristle, piece of toothpick or fish bone, for example; or it may be caused by abuse with floss silk or too hard a toothbrush used incorrectly. A papillitis will result, that is, an acute inflammation of a papilla. The cause should be found early and eliminated.

Gingivitis associated with hormonal changes.

It is well known that during adolescence, sometimes during menstrual periods and in pregnancy gingival disturbances do take place. There is no doubt that alterations in the tissue tone due to hormonal changes may be an underlying factor in these gingival disturbances but there is also in most cases other factors such as local irritation and lack of oral hygiene. Therefore it is more than ever important during these conditions to pay attention to oral hygiene and massage to counter any tendency to lost tissue tone. If this be done the gingival margins can be kept normal, and conversely, if this be not done, then, for example at the termination of the pregnancy, the gingivitis will still persist, proving the existence of local factors. So it

Fig. 12.—Thickened periodontal membrane at incisal aspect of $\underline{|1}$. Tooth in traumatic occlusion.

is not enough to tell a pregnant patient that nothing need be done about her gingivitis as it will disappear at parturition because this will probably not be the case, unless there is due attention given to oral hygiene.

Before leaving the inflammatory conditions the writer would like to point out that there are certain investigators who think that even in cases like diabetes and leukaemia where characteristic lesions occur, there is always an underlying picture of local irritation such as calculus and lack of oral hygiene. Be this as it may, it further emphasises the role of oral hygiene in preventing periodontal disorders.

Evidence of early changes due to traumatic occlusion (Fig. 7).

One tooth or several teeth will be seen to take the greatest part of the occlusal stress in certain movements of the jaws or in centric occlusion. There may be signs of gingival recession in teeth suffering traumatic occlusion or the anterior teeth may show much incisal wear while the cusps on the molar teeth are very high. The x-ray will show a thickened periodontal membrane in these cases (Fig. 12). All these signs should be noted and the occlusion corrected to prevent serious damage to the periodontium. All bad habits which tend to produce traumatic occlusion should be eliminated, for example, opening bobby pins with the teeth.

Fig. 13.—Toothbrush abrasion.

Toothbrush abrasion.

This should be corrected as soon as it is noticed to prevent serious damage to the teeth. Where this is occurring there will be loss of tooth structure and gingival tissue on one or both sides of the mouth or around one prominent tooth only (Fig. 13). The patient's brushing methods should be checked and corrected.

The writer repeats and stresses that, in all these cases of marginal gingivitis or where there is some departure from the normal gingival picture (and these are probably seen daily), the obvious should be attended to first, that is, attention to all phases of oral hygiene as outlined earlier. If this fails then other phases of periodontal treatment must be instituted, and then this again must be followed by adequate home care.

Gottlieb and Orban[6] sum up the whole status of oral hygiene when they say that no attempt at diagnosis should be made in the case of a chronic gingivitis in a mouth that has been grossly neglected until a thorough prophylaxis has been carried out; then with home care it may very often be seen that the condition no longer exists.

The object of periodontal therapy as pointed out by Goldman[7] . . .

. . . is to produce firm, pink, well attached gingivae with no crevice extending beyond a normal depth. There should be good gingival architectural form. The teeth should be firm in their sockets without tenderness or movement. Radiographically an intact lamina dura should be present. There must be no focus of infection or inflammation.

The writer, however, believes it is not sufficient merely to produce this end, but a constant vigilance and effort by dentist and patient must be maintained to keep the tissues in this state. In conclusion, the writer would like to quote from Kirkland[8] about the maintenance of the health of the periodontal tissues after treatment of periodontitis and of course it applies equally well to these cases of marginal gingivitis the writer has discussed.

The gingivae must be kept under constant observation by following the same routine as that commonly used to control caries. By this we mean that the mouth cannot be treated and the patient permanently dismissed when the wounds are healed, as may be done in cases of removal of an appendix or in tonsillectomy. On the contrary, the gingival tissues must be watched for recurrent gingival inflammation with the same meticulous care as is observed in the management of caries. When a new pocket does occur, it is just as important to give it prompt treatment as it is to fill a new cavity in a tooth and it should be done as soon as possible after it is discovered.

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PART II.

MALOCCLUSION.

G. Morse Withycombe, B.D.S. (Syd.)

To date there has been found no one, initial causative factor in Periodontitis or Gingivitis. It is only by taking each case on its merits, bearing in mind all the factors we know and treating all phases of the disease as they present, that we can hope for any successful and permanent results to our efforts in treatment.

The resources of all branches of dentistry—Radiography, Oral Hygiene, Pedodontia, Orthodontia, Operative, Prosthodontia and Exodontia — must be considered and drawn upon, when undertaking the prevention and treatment of disorders of the periodontium.

The practitioner must learn to recognise the normal and abnormal in occlusal relationships, and no attempt should be made to interfere with the status quo unless an improvement can be anticipated. The ideal is to distribute the forces of mastication over as great an area as possible. An estimate can usually be made of the degree of occlusal wear that normally should have occurred for the particular patient up to that time, and care must be exercised not to exceed that limit of wear by excessive and optimistic grinding.

Generally speaking for the gingival and periodontal tissues to be healthy the teeth should be in normal physiological function. The gingival and periodontal tissues receive stimuli from mastication, and depending on the state of the occlusion, these stimuli will be either good, bad or indifferent.

For the purpose of this paper the three most common forms of malocclusion met with will be discussed:

(1) Antero-posterior malocclusion.

(2) Uni-lateral malocclusion.

(3) Vertical malocclusion.

(1) *Antero-posterior malocclusion.*

An antero-posterior or protrusive action in mastication is perhaps the most common seen today; it is not in the scope of this paper to go into reasons for this but mention must be made of the almost universal desire today to have all food tender or pre-cooked and to consume it in the shortest possible time. Some patients believe that to swing the jaw laterally and chew vigorously is actually "vulgar," but surely such a normal action, although somewhat uncommon, is not vulgar.

With the protrusive masticatory action, labio-incisal wear will be noticed with corresponding lack of wear on the posterior cusps; clinical and radiographic evidence of trauma and marginal bone loss would be expected in the anterior region; the tissue tone of the posteriors would tend to be poor, due to lack of function, and the degree and plane of incisal wear would be indicative of the degree of overbite present. The degree of lateral movement present usually decreases in relation to the age of the patient: many adults will be found locked in centric, with evidence of atrophy of the lateral movement musclature, due to disuse.

In adolescents, cases will be seen where, although the masticatory action is obviously antero-posterior, there is evidence of previous

bi-lateral wear. In these cases note should be taken of the presence of third molars which have erupted late or out of normal arch alignment, and have interfered with or prevented the previous bi-lateral occlusion. Antero-posterior masticatory action may also be due to the effect of badly decayed or missing posterior teeth, with the consequent interference from the exfoliating opposing tooth and drifting of the adjacent teeth. These circumstances may often be seen as a reason for the change from a lateral to a protrusive type of mastication. The assistance of the Orthodontist or Prosthodontist is imperative here and one can put in a plea for restoration of missing teeth to complete the arches. The injury caused by lack of contact points due to drifting and exfoliation of the adjacent or opposing is far too common and unnecessary; it is the dentist's right and duty to restore fully his patient's dentition.

(2) *Uni-lateral malocclusion.*

Mastication on only one side of the mouth: once again I feel that the commonest reasons for this restricted action are: (a) painfully carious or missing teeth in the posterior region and (b) the soft type of food eaten, requiring little masticatory force, especially at the adolescent age when everything is done in a hurry, including eating.

I have found in practice that easily 50 per cent. of patients, who show any degree of eccentric occlusion, are uni-lateral, usually the left side. The fact of their being right or left handed seems to bear no relationship. The tissues on the left side would be expected to show a greater degree of tissue tone, due in part to normal exercise and also to the fact that the left side in cleaning, probably through ease of access, receives more attention. Clinically and radiographically the opposite side will be found to have less tissue tone and greater marginal bone absorption. The cusps show definite wear facets, especially in the cuspid and bicuspid regions, depending on the degree of antero-lateral movement present.

In balancing this occlusion the greatest difficulty is often to persuade the patient that the mandible will in fact, move to the required eccentric position. Home practice and the use of chewing gum is often necessary. It must be remembered that the muscles involved are, according to the age of the patient, to some degree atrophied, and will need patience and exercise to acquire some degree of flexibility: these remarks will of course apply in most cases where adults are concerned.

(3) *Vertical malocclusion (a chopping bite).*

In this type we have the true centric action with no lateral or protrusive movement. The facets are worn in an inciso-gingival direction, the length of the facets depending on the amount of the over-bite and the degree of inter-digitation present. This very often will be particularly noticeable in the bicuspid to bicuspid area. Even if the posteriors are still standing despite the good resistance to the vertical load, evidence will be seen of food impaction areas both palatally and interproximally. In some cases where food impaction occurs, poor operative work is blamed for faulty contacts, when in fact the operative restorations have been adequate, but insufficient attention has been paid to the wedging action from the opposing cusps. In other cases where food impaction occurs a prosthetic appliance is worn that confines the mandible into vertical movements only; and here again we have an instance where the periodontal aspect is intimately related to other branches of conservative work.

A prosthetic appliance of the stress breaker type which distributes the load over many teeth and the greatest possible area of soft tissue, but free of the palatal and lingual gingivae, is desirable for periodontal and gingival health. Wherever possible, the fixed bridge is the restoration of choice; constructed on the stress divider principle, it enables physiological movement of the abutment teeth in function.

It is good practice to balance the occlusion before constructing any prosthetic appliance. Not only will this save the operator endless hours in making dentures comfortable, but it will also save the denture or pontic from being so "adjusted and eased" that it ultimately bears little resemblance to its original design and form.

Too often we see a natural bi-lateral dentition locked in centric by the mal-occluding denture cusps. If the patient uses any degree of lateral movement, the clasped teeth must inevitably be in hyperfunction and eventually collapse under the strain.

Mention must be made in this section of several habits that are trauma producing, viz. cotton biting, bobby-pin opening which is much commoner, and even more destructive, and lastly nail-biting. An interesting observation of nail-biting is that very often a Vincent's infection, particularly obstinate in one region, will be found to be due to the constant irritation and trauma produced from nail-biting in this area. A constant gnawing action in one area will cause not only inflammation of the periodontium, but rotation, mobility, and eventual loss of one or more teeth as a direct consequence.

Balancing the occlusion.

The mechanics of occlusal balancing of the natural dentition varies considerably from that of artificial dentures.

It must be remembered that the mandible can make movements eccentrically far beyond the scope of any anatomical articulator. Prior to grinding in the occlusion an articulated pair of models for instruction in jaw movements will be found very helpful, as to correct a patient's occlusion without his co-operation is well nigh impossible.

Time is always well spent in ascertaining the true functional centric and eccentric movements. When the centric is decided upon the teeth are ground so as to bring as many of the teeth as possible into contact in this position, with the incisors free of contact except with pressure. It should be possible to pull a thin strip of cellophane between the eight incisors to test this.

Note is taken of the areas in contact in centric, as care must be taken to lose only the minimum amount of vertical dimension.

The correction of one lateral excursion is attempted and carried through at one sitting. The mandible is moved laterally several times until a comfortable and even movement is obtained. It should be noted whether the action is true lateral, postero-lateral or antero-lateral; a good average working excursion is half the width of a second bicuspid tooth bucco-lingually. Any interference from the palatal cusps of the opposite side is freed at this stage and checked continually. Grinding is then carried out on the functioning side to produce a plane on the appropriate tooth that will free the excursion and still work in with the planned and ultimate occlusion.

Retention of as much cusp formation as possible is desirable from the aesthetic viewpoint, vertical dimension, extent of lateral movement desired and the shearing action of mastication.

The use of pencil carbon paper, while perhaps slower, is more exact than the usual double-sided articulating paper, as it will mark one jaw only, except under heavy pressure in the final adjustments, when the colour will be carried through to the offending high spots. The use of small diameter and narrow edge diamond stones with a warm water spray makes the work quicker and more precise for the operator and more pleasant for the patient.

The practice of holding the chin in the fingers with the index finger lying along the body of the mandible externally during lateral excursion will be found, especially in the later stages, to assist in guidance and feeling the freedom of movement or interference from cusps of the opposite side. These can often be detected before the patient is aware of them.

In balancing a cross bite the procedure is similar, except that the balancing bite becomes the working bite; that is, with the mandible in left lateral function the right side is in working occlusion. The degree or extent of lateral movement in these cases is usually less than in normal excursions.

The ideal protrusive balance, with the eight incisors in contact and at least one posterior contact on either side, is very rare. To obtain this in the average mouth would necessitate radical grinding of the incisors or posteriors. In practice it is found that to distribute the load over as many incisors as possible commensurate with the aesthetics gives a good working result. Finally, at the end of every session the centric is checked for any excessive weight on the incisors at rest.

In conclusion one should be mindful of these four points:

(1) Any drastic change in occlusal function must be made with the full understanding and co-operation of the patient before and after treatment.

(2) The balancing of the occlusion is an essential part of the complete treatment for gingival and periodontal disorders.

(3) Radiographic interpretation and assessment is necessary before any change in occlusal loading is made.

(4) Physiologic function is essential for the health of the periodontium.

PART III.

SURGERY IN PERIODONTAL TREATMENT.

Ralph Tompson, B.D.S. (Syd.)

There are several forms of surgery employed in the treatment of periodontal disease. These include gingival resection (or gingivectomy), electro-surgery, electro-coagulation, thermal cauterisation (by means of the electric cautery), chemical cauterisation (by the use of escharotics), and the so-called "gingival flap" operation.

Referring to these various procedures in reverse order: the gingival flap operation may be described as a surgical aid to the "conservative" method of treatment, the principle of which is closure of the pocket by re-attachment, that is, by producing a union between the cementum and either epithelium or connective tissue. The sole function of the gingival flap operation is to facilitate the preparation of the pocket to that end.

The re-attachment technique is not widely practised in Australia; and since the whole question of re-attachment is highly contentious, it is not proposed to discuss it in this paper.

Chemical and thermal cauterisation are employed to achieve the same result as can be more efficiently obtained by gingival resection, and therefore their use is not favoured.

In electro-coagulation, which is achieved by means of a high-frequency current, the tissue is not actually cauterised, but its protein is coagulated. This causes necrosis, resulting in sloughing and separation of the unwanted tissue. The method has some merits, but is seldom preferable to the use of the knife.

Electro-surgery employs a high-frequency current of a somewhat different character, which causes the electrode to actually cut through the tissue, thus making possible its complete removal at the time of the operation. At the same time the surface of the remaining tissue is coagulated. I have not yet used this method, but it would appear to have two possible advantages over the knife: it may be psychologically less distasteful to some patients, and it is said to produce no bleeding, thus facilitating the operation. As against these advantages, it produces an odour of burning flesh, which may well be very objectionable to many people.

GINGIVECTOMY.

The operation of gingivectomy, performed with the gingival scalpel, has been well tried and proved successful as a means of eradicating the periodontal pocket. The idea that a gingivectomy *alone* will permanently cure periodontal disease is, however, entirely erroneous. Unfortunately, it is a not uncommon practice to treat a case of pyorrhoea by surgery alone, the patient then being dismissed with a somewhat vague admonition to massage the gums in future, but without any detailed advice or instruction in the precautions necessary to prevent recurrence of the disease.

In point of fact, gingival surgery constitutes *only one phase* of a planned system of treatment—one important link in a chain whose other links are equally essential. Basic requirements which must always be met include the prior elimination of acute infection (if present) and acute or sub-acute inflammation, complete removal of calculus (both supra- and sub-gingival), adjustment of occlusion where indicated, correction of faulty restorations, attention to diet if necessary, and education of the patient for purposes of co-operation. Detailed discussion of these requirements, however, is outside the scope of this paper.

The objects of the surgical operation itself are: (1) to completely eradicate all periodontal pockets and (2) to provide a gingival form, or contour, which will enable the patient to achieve a high standard of hygiene and gingival stimulation in order to *maintain* a state of periodontal health.

Indications for gingivectomy.

This operation is employed: (1) in cases of chronic gingivitis (with or without an early degree of bone-resorption) where conservative methods of treatment have failed; (2) when a more advanced degree of bone-resorption indicates the uselessness of attempting treatment by conservative measures; (3) with or without bone-resorption, when a true gingival hyperplasia has occurred (as distinct from the oedematous swelling of the gingival margins which usually accompanies chronic gingivitis); (4) often following Vincent's infection, in order to ensure against recurrence by eradicating possible incubation areas. (See figures 1-3.)

Examples of hyperplasia include the common form which frequently results from chronic gingivitis of long duration; hyperplasia of the labial gingiva resulting from mouth-breathing; that occurring in some children at puberty; a specific form (very fibrous) resulting from dilantin-therapy in the treatment of epilepsy; and in many cases of pregnancy gingivitis.

The usual conservative methods of treatment, directed at improving the standard of oral hygiene, gingival massage, and removal of sub-gingival calculus should be sufficient to clear up a case of pregnancy gingivitis unaccompanied by hyperplasia; but when the latter has occurred, resection of the hyperplastic tissue is indicated[1].

Resection of the hyperplastic tissue resulting from dilantin-therapy should be limited to those cases where it is so severe as to interfere with the patient's ability to masticate food, or where the presence of this tissue precludes the possibility of restoring carious teeth, since continuance of the administration of dilantin will inevitably produce a recurrence. For the same reason, caution should also be observed in the treatment of hyperplasia associated with puberty, until such time as this period has passed. Hyperplasia caused by mouth breathing, on the other hand, can usually be satisfactorily dealt with by surgery, since in these cases the usual methods of post-operative stimulation of the gingival margins will prevent recurrence.

Prognosis.

Radiographs are of great value in ascertaining the degree of bone-resorption and assessing the possibility of eliminating the existing pockets, although it must be remembered that they cannot be relied on as a measure of the actual *pocket-depth*, since the gingival margin may have moved considerably towards the

root-apex (through recession) or towards the occlusal surface (through oedema and/or hyperplasia). Under such circumstances the degree of bone-resportion and the clinical depth of the pocket do not coincide. X-rays are especially valuable where the bone-resorption is advanced; in fact it may be said that in such cases a reliable prognosis cannot be made without them. In short, the x-ray should be regarded as an indispensable complement to the detailed clinical examination, which, however, it must never be allowed to replace.

Another important form of examination when making one's prognosis is a careful investigation of the stability of the teeth. The degree of mobility of each tooth should be accurately recorded, and considered carefully when estimating the life-prospect of individual teeth and of the dentition as a whole. In this connection it may be said that a certain degree of mobility in the incisor teeth — especially the lowers — is not as serious as an equivalent mobility in the canines, bicuspids, and molars, since the amount of stress borne by the incisors is seldom as great as that taken by the other teeth, and can often be greatly reduced by judicious grinding — a thing not always possible to attain elsewhere.

If partial dentures are required, the stability of the teeth must be very carefully considered, with special reference to the stresses and strains which they will be called upon to take. Seriously loosened teeth deprived of much of their original bone-support, even if not intended to act as stabilisers, should be extracted; while, of the remainder, only the really firm teeth should be used to actually support the denture.

A thorough examination must always be made of the posterior teeth in reference to possible involvement of their bifurcations and trifurcations. If this has occurred, the tooth concerned should be condemned, except in those cases of very early involvement in which the patient's ability to keep the opening clean is pretty well assured. This is seldom possible on the mesial or distal of an upper molar when the adjacent tooth is present.

It is usually advisable to perform the necessary extractions before attempting treatment of the remaining teeth by gingival resection.

An important consideration which must not be overlooked is the possible effect of the operation on the patient's appearance. Fortunately this problem seldom arises; but in cases where, because of an exceptionally high lip-level, elimination of periodontal pockets surrounding the upper anteriors is calculated to produce an unsightly result, consideration may have to be given to artificial replacement of the teeth concerned.

Surgical treatment-planning.

Having decided that a gingivectomy is necessary, and having followed the lines of treatment described in Parts I and II of this paper, to the point at which the operation may be suitably performed, the first step is to determine exactly where and to what depth the gums should be resected. In certain parts of the mouth there may be a complete absence of pockets, while in other places there may be considerable variations in depth; hence the necessity to make a thorough clinical examination of the entire gingival margins, with the aid of a graduated pocket probe, and to record the findings clearly and accurately on the patient's treatment card. A card which includes the usual illustrations of the complete dentition is best for this purpose.

This detailed record is better obtained at this stage than at the initial examination, since the pre-surgical methods of treatment already employed may well have succeeded in eliminating any shallow pockets resulting entirely from oedema of the gingival margins.

A suitable section of the mouth should be selected for operation, its extent depending partly on the depth of the pockets to be eradicated. Generally speaking, the ideal procedure is to treat as large an area at the one session as is possible without causing the patient too much distress during the operation or too much pain or discomfort afterwards. Usually post-operative pain is very slight or altogether absent, but it is naturally more likely to occur after excision of deep pockets, necessitating the removal of a large amount of tissue and the exposure of an unusually large area of raw tissue (as in the case of deep pockets on the palatal surfaces of the teeth). Therefore, if the pockets are very deep, it may be considered desirable to limit the area of treatment at one operation to a greater extent than would normally be considered necessary.

Assuming that the entire dentition is involved, and that the pockets are generally of moderate depth, it is reasonable to divide the mouth for the purpose of operation into three sections. These sections could comprise, for example: (1) upper right and upper anterior; (2) upper left and lower left; and (3) lower anterior and lower right. In this way the patient's ability to masticate food while the dressings are in place is not too greatly impaired, post-operative pain should not be

Fig. 1.—Chronic hyperplastic gingivitis accompanied by oedema. Restoration to normal gingival form could be achieved only by surgery.

Fig. 3.—Vincent's Infection. Following elimination of infection, normal gingival contour will have to be re-established by surgery in order to make possible a high standard of hygiene of the interdental spaces.

Fig. 2.—In contrast with case shown in figure 1, the swelling adjacent to the right lateral is due not to fibrous overgrowth (hyperplasia), but solely to oedema accompanying the gingivitis. Restoration to normal contour could be secured by gingival massage.

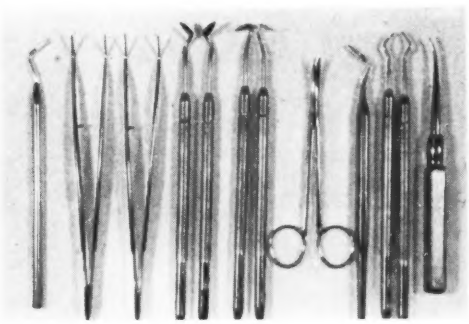

Fig. 4.—Instruments for use in gingivectomy. Left to right: Pocket probe graduated in millimetres; pair of instruments (right and left) for marking externally the base of the pocket; two types of blades (each pair in right and left forms) suitable for gingival resection throughout the mouth; curved gum-scissors; fine tapering file for removal of granulations from interdental spaces; pair of contra-angled scalers; narrow-edged, hand pressure bone chisel for occasional use (see text under heading "Surgical Technique").

Fig. 5.—Case requiring surgery for elimination of pockets due to moderate degree of bone resorption, immediately after removal of surgical dressings.

Fig. 7.—Case involving advanced bone-loss.

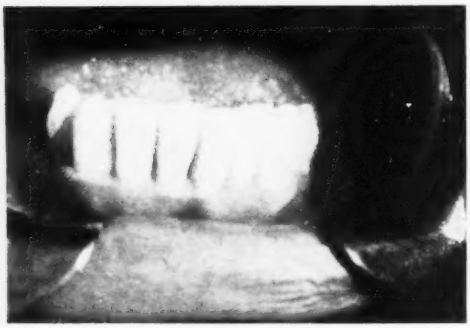

Fig. 6.—One week later.

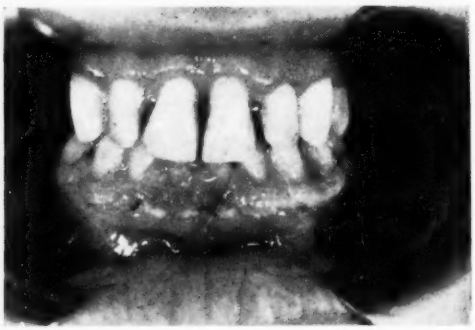

Fig. 8.—Same case after treatment by gingivectomy—16 days after removal of dressings. Surgical dressings in place following gingivectomy in lower jaw.

severe enough to matter seriously (indeed it may not occur at all), and yet the patient is spared the unpleasantness of having to submit to numerous operations, as would be necessary if only three or four teeth were treated at a time. If, on the other hand, the pockets are deep, one quarter of the mouth may represent a reasonable limit — perhaps even less.

The patient's temperament is another factor which of course should not be overlooked. One would hesitate to attempt as much for a nervous, highly-strung patient as for a placid one — though nervous people are obviously the very ones to whom the prospect of facing repeated operations is most distasteful. For such people, premedication with NEMBUDEINE or some similar sedative drug is of great assistance.

Equipment.

Equipment required for this operation (apart from that needed to obtain anaethesia) comprises: suitably shaped knives; a pair of slender curved scissors; a pocket-probe; an instrument for marking externally the position of the pocket-base; a suitably shaped pair of scalers; a file or curette for removing granulation tissue; sterile swabs; and zinc oxide-eugenol paste, wool, and dental floss for preparation and application of surgical dressings. An aspirator for removing blood and saliva is an added advantage. (See figure 4.)

Anaesthesia.

The best way to obtain local anaesthesia is by injection into the base of the interdental papilla, as this method controls bleeding during the operation better than any other. For the same reason, an anaesthetic solution possessing a strong haemostatic action should be chosen.

Surgical technique.

Having decided on the area to be treated, and having obtained anaethesia, the x-rays and the relevant notations are studied with reference to determining the point of insertion of the scalpel. The final guide in making this decision is, of course, the pocket-probe; or better still — especially where the pocket is deep — a special instrument for marking the external gingival surface at the point opposite the base of the pocket. This instrument consists of a pair of stainless steel tweezers with long, very slender beaks, the tip of one beak being fitted with a short sharp point, directed at 90° towards the opposing beak. The plain beak is inserted to the base of the pocket and the tweezers closed, resulting in a slight puncture of the gum, which leaves a tiny surface mark representing the level of the pocket-base. This indicates the point for insertion of the knife — at least where the tissue to be excised is thin, as on most labial and buccal surfaces. Where it is thick, as in the case of the palatal surfaces, it is necessary to start the incision at a point slightly apical to the pocket-base (that is, apically to the puncture-point), since it is very important to finish the operation with a correctly contoured gingival margin: in other words, with a natural slope towards the neck of the tooth. Excising the gum at right angles to the axis of the tooth often results in a broad shelf of tissue, which will make it much more difficult for the patient to keep the gingival margin at this point clean and adequately stimulated.

The blade, which must have a razor-keen edge (and which is best sharpened by a surgical cutler) is inserted at the correct slope until it reaches the surface of the tooth. It is then drawn across the pre-determined line of incision until the limit of the area to be treated is reached. This is usually done first on the lingual or palatal side, being followed by a similar operation on the labial or buccal side. In practice, unless a series of puncture-marks has been made as suggested, it is wise to cut only a small section at a time, interrupting the procedure at short intervals in order to check, with a probe, the exact level at which to insert the knife for the next section.

When the incision is complete, the excised tissue can be lifted away from the teeth with suitable instruments, such as a pair of dissecting forceps or a pair of "Morse" scalers nos. 4 and 5. The tissue to be removed should be found completely detached, provided the knife has been carried right through to the tooth and well into the interdental spaces from both sides. However, it is not always easy to achieve complete excision in the more confined interdental spaces; in such cases it may be necessary to insert the knife again before the excised tissue can be lifted away as desired.

If the knives used are sufficiently sharp, a generally clean, smooth wound should result. If, however, the surface is not perfectly smooth, any fragments of tissue responsible can be removed with a fine pair of gum-scissors. Small tags of granulation tissue remaining in the interdental space are best removed with fine scalers or curettes, which are pressed firmly against the surface of the tooth; or in some cases by means of a special interdental file. All granulation tissue *must* be removed.

Any interference with the supporting bone is contra-indicated, except in rare instances. It used to be thought that periodontitis involved necrosis of the alveolar bone, giving rise to the old term "Pyorrhoea alveolaris;" but this theory is no longer considered valid. According to Gottlieb, Fish, and others, bone-resorption in chronic periodontal disease results from absorption of bacterial toxins, not from actual infection, since the bacteria themselves do not invade the bone. Once the source of toxic absorption is eliminated, any existing osteitis will disappear and the bone remaining around the tooth will return to a state of health. Therefore all bone-support remaining to the tooth should be conserved, and the practice of curetting the alveolar crest during a gingivectomy must be severely condemned. The object of a gingivectomy should be to remove all detached gingival tissue from around the tooth, without interfering with any of the tissues that are still attached. All attached alveolar crest fibres and transverse fibres should be retained, thus leaving the alveolar bone completely covered.

Occasionally, however, resorption of the centre of the alveolar crest may be found to have occurred to a greater extent than on its lingual or buccal aspects. In such cases, removal of the latter portions of the alveolar crest is not only justified but actually necessary. Only thus can the pocket be completely eliminated and the patient enabled to keep the interdental spaces thoroughly clean and to adequately stimulate the whole of the interdental tissue. This levelling-off of the interdental bone can be effected by the use of files or very slender bone-chisels (about 1 mm. in width).

If the incision was made at the correct level, no vestige of the pockets will now remain. It should never be *assumed*, however, that this result has been achieved; in order to make quite sure, the whole of the gingival margin around every tooth in the area treated should be carefully examined with a pocket-probe, and any shallow crevices still remaining eliminated by further excision of the tissue concerned.

It is unlikely that all the sub-gingival calculus will have been removed during the preliminary stages of treatment prior to the gingivectomy, if the pockets were at all deep. Although the residue need not be wholly removed at this stage, it is desirable to get rid of at least that portion which is actually in contact with the edge of the wound, in order to prevent the possibility of irritation of the latter during healing.

Application of surgical dressings.

Finally, surgical dressings are applied to the wound, in order to protect it against irritation and infection, to control pain, and to prevent haemorrhage. It is usually desirable to leave them in place for seven days.

The dressings consist of a mixture of eugenol and zinc oxide incorporated in a material which will give it sufficient strength to remain securely in place for the prescribed period. Experience has shown that there is no need for antiseptics, astringents, or haemostatic agents. (Eugenol itself is a mild antiseptic.)

I have used the term "dressings" in preference to the more popular term "packs" because I believe the latter to be a misnomer, seeming to imply that the dressings are "packed" into place under pressure — a most undesirable practice. The dressings should be applied gently, being pressed into place just firmly enough to mould them into conformity with the gingival contour around the necks of the teeth. They should completely cover the entire wound, but should not extend beyond its edge.

There are several ways in which dressings may be prepared, but the one which I greatly prefer employs two lengths of (autoclaved) knitting-wool impregnated with the zinc oxide-eugenol paste. These are placed on opposite (labial and lingual) sides of the teeth and bound in place with dental floss, which is passed under and over the wool in the following manner. The floss is first tied to one end of one of these prepared lengths of wool (say the one to be applied to the labial side), and then passed through the interdental space at one end of the group of teeth concerned, thus drawing the attached end of the wool into the labial embrasure. One end of the other length of wool is then laid across the floss, which is now on the lingual side, and the floss is brought back again through the interdental space, thereby drawing the second length of wool into the *lingual* embrasure.

The labially placed wool is now laid across the floss, which is then passed through the next interdental space, thereby drawing the wool into the second embrasure. The action is repeated on the lingual side, bringing the floss through the third interdental space and the lingual wool into the third embrasure. This under-and-over action is continued until the last interdental space is reached. Here the floss is carried through and back again, as at the beginning, after which the surplus wool and floss are cut off.

A portion of the prepared wool having been previously cut up into small pieces, these are then inserted in each of the interdental spaces from both labial and lingual sides, in order to fill them, after which the whole dressing is moulded into shape with the aid of a gauze pad.

This method results in a remarkably secure dressing, the advantages of which are considerable: (1) it is almost impossible to dislodge during mastication; (2) because it is so firm, there is no movement whatever to irritate the tissues—a very common source of pain; (3) close adaptation of dressing to tissues is maintained throughout, thus preventing the formation of unwanted granulation tissue under the dressing during healing — a frequent source of trouble, if any lifting of the material occurs; and (4) it provides a particularly neat form of covering.

If the operation has been correctly performed in every detail, and the dressings suitably applied, in most cases there will be no post-operative complications beyond a mild soreness, though after eradication of very deep pockets the pain is occasionally a little greater. In a large proportion of cases there is no pain whatever.

Removal of dressings.

After one week the dressings are removed. This can be done fairly easily with a suitable instrument, such as a pair of Morse scalers nos. 4 and 5. These are sturdy, contra-angled scalers, designed for the removal of gross calcular deposits.

The gingival surface should present a smooth, half-healed appearance. There should be no granulations or tags of incompletely severed tissue to interfere with the completion of a rapid and uneventful healing, which should take place by first intention.

The only operation that should be necessary on removing the dressings is the dislodgement of small fragments of what was previously sub-gingival calculus — or at least any particles in contact with the new gingival margins; calculus remaining in places coronal to the gingival margins may be left until later, if desired, since it will not interfere with healing. (See figures 5-8.)

Post-operative care.

It is at this point that the co-operation of the patient, based on the advice and instruction already received, becomes *and remains* essential to the success of the whole treatment. He should be instructed to begin immediately the routine of interdental stimulation with balsa-wood sticks, and to massage the gingival margins with the toothbrush as soon as possible.

An inspection should be made of the treated area at the expiration of two to three weeks, when healing should be complete, though in some cases it may take a little longer before keratinisation is sufficiently advanced to enable the tissues to be vigorously massaged without causing slight discomfort. At this appointment the patient's home-care should be checked.

Also at this appointment, or as soon as the first section of the mouth has healed to the satisfaction of the operator, the next section is treated — and so on until all pockets have been eliminated.

Before finally dismissing the patient, all remaining traces of calculus are removed and the teeth polished. An arrangement should be made to recall the patient at regular intervals for thorough inspection of the gingival margins, removal of calculus, and to check the standard of home-care if necessary.

PERIODONTOSIS.

The foregoing rationale of treatment is recognised as being applicable to periodontal disease of local inflammatory origin (periodontitis), but it may not be generally realised that it is also efficacious in cases of periodontosis.

This disease is usually considered to be of systemic origin, being variously attributed to dietary deficiencies, metabolic disturbance, endocrine dysfunction, blood dyscrasias, etc., while Orban has recently suggested the possibility that periodontosis may be due to a developmental fault in the epithelial network of the periodontal membrane. The consensus of opinion, however, is that the aetiology of periodontosis is unknown, and for this reason it is generally considered to be incapable of treatment.

Nevertheless, despite this lack of knowledge of the cause of periodontosis, experience has shown that, if intercepted in time, its effects can be successfully treated by the foregoing methods, and that in most cases the disease can then be satisfactorily controlled by the usual methods of home-care.

This opinion is supported by Glickman[2], who says:

> The theory that prompts persistent effort to isolate a "systemic" type of periodontal disease is presumptive. While it seeks for the mechanism of systemic participation in one type of case, it automatically assumes that it is entirely absent from others. Cases of periodontal disease differ from one another not in the complete absence or presence of systemic influence, but rather in the degree of systemic participation. There is systemic participation in *all* cases of periodontal disease.
>
> With respect to treatment, it is important that one should not minimise the contribution of *local* influences in that type of periodontal destruction resulting *primarily* from *systemic* influences. Elimination of the local factors contributing to the destructive changes often effects a clinical control of the disease even though destructive tendencies of systemic origin may still exist.

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Preventive Periodontia for the General Practitioner*

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There is no need to stress the almost universal incidence of periodontal disease[1] and the fact that, in the eyes of most patients and their dentists, the only treatment is the extraction of the teeth. This situation has arisen because dentists have been pre-occupied with dental caries and have largely ignored periodontal disease. One reason for this is the fact that dental caries causes pain while most periodontal disease takes the form of a chronic degeneration which develops so slowly that the patient is often unaware of the severity of the periodontal involvement until conservative treatment is no longer possible. If we continue the comparison with dental caries, we will agree that treatment of periodontal disease is not suggested until a stage is reached corresponding to death of the pulp instead of instituting periodontal treatment at the stage corresponding to prophylactic odontomy.

Both diseases are accompanied by a tissue breakdown; but, while we can replace tooth substance satisfactorily, there are definite limitations to our treatment of periodontal pockets and abnormal mobility. For this and other reasons, control of periodontal disease depends on the elimination of causes and early treatment. Unfortunately, dentistry as practised by the majority falls far short of this ideal and we find that dentists are responsible, either directly or indirectly, for a considerable amount of the periodontal disease in the mouths of their patients.

This situation is a reproach to Dentistry and calls for a re-orientation by dentists of their approach to the practice of their profession.

Two factors may force re-orientation sooner than we think—one is the ageing population and the other is the prospect that public health measures may so lower the incidence of caries that dentists will be able to keep it under control.

Prevention of periodontal disease by the general practitioner depends on the early recognition of the signs of periodontal disease and on an understanding of the many etiological factors involved and their complex interrelationship. Box[2] has classified the signs of incipient periodontal disease under eight headings and, of these, the most important are abnormal mobility and changes in the texture, the colour and the contour of the gingivae. Many of the etiological factors can be eliminated and others can be made innocuous by increasing the local and general resistance, and therefore we should tell our patients that the periodontal tissues of the majority of people who have good occlusion and no systemic disease can be kept in a healthy state if we and they apply what is already known[1]. For the remainder, a breakdown of the periodontal tissues can be deferred if dentist, physician and patient all do their part. There is general agreement that local irritants such as calculus, food debris, faulty restorations, mouth breathing, food impaction, etc., act as exciting causes of periodontal disease and although there is not unanimity about the role of abnormal function "a considerable preponderance of evidence supports the conclusion that occlusal trauma should be accepted as a factor in periodontal disease[3]." Lack of normal function is also a factor in lowering the resistance of the periodontal tissues[4].

The part played by systemic factors is not easy to assess. It is evident that there is no one factor common to all periodontal disease[5] and the relative importance of any single factor may be influenced by so many other factors. Glickman has put forward his "Bone factor concept[6]" and has discussed it in the light of Selye's "General adaption syndrome[7]." Others who have discussed the complex inter-relationship between the various factors concerned are Gratzinger[8] and Boyle[9].

It is generally agreed that systemic factors may affect the timing of the appearance of periodontal disease, its rate of progress and the prognosis for treatment but, as general practitioners endeavouring to prevent periodontal disease, we must resist the temptation to become so interested in a search for obscure systemic factors that we overlook or neglect the obvious local factors. With the possible exception of true periodontosis, periodontal disease never occurs in the absence of local exciting factors. Where there are no obvious systemic factors, a good working rule is to eliminate *all* local causes of periodontal disease, instruct the patient in proper home care and, if the tissues do not respond as expected, suspect the presence of a systemic factor, and arrange for a complete medical examination and all necessary special tests, including diet analysis.

Every dentist should be familiar with the more obvious oral manifestations of systemic disease and he should appreciate the importance of optimum nutrition and the various factors, besides the diet, which affect nutrition, but he should not attempt to usurp the

* Read at the 13th Australian Dental Congress, Brisbane, June, 1953.

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functions of the patient's physician. He should concentrate on the elimination of all local irritations, the instruction of the patient in home care and the establishment of normal occlusal function. Most cases of periodontal disease could have been prevented if this had been done at the appropriate time. It is the general practitioner's responsibility to do these things — no one else can. We will now consider each of these three responsibilities in turn.

As the chart (Fig. 1) shows, the prevention of some forms of local irritation and the elimination of others are important responsibilities of every general practitioner.

PREVENTION OF PERIODONTAL DISEASE.
THE GENERAL PRACTITIONER HAS THREE RESPONSIBILITIES

PREVENT LOCAL IRRITATIONS
1. Calculus and food debris.
2. Food impaction.
3. Faulty restorations or operative technique.
4. Mouth breathing.

INSTRUCT IN HOME CARE
1. Natural and artificial oral hygiene.
2. Toothbrushes, dentifrices, special aids.
3. Methods of toothbrushing.
4. Teaching the patient.

ESTABLISH NORMAL OCCLUSAL FUNCTION
METHODS: 1. Orthodontic treatment (if feasible).
2. Extraction of malposed, extruded or impacted teeth.
3. Use of partial dentures, bridges, onlays, etc.
4. Occlusal equilibration by grinding.

Calculus and food debris are the most common and are an indication of poor oral hygiene. Because many patients think that calculus formation is inevitable, it should be made quite clear that calculus cannot form on a clean surface and that the control of calculus formation is in the patient's own hands. Existing deposits must be removed with scalers and curettes, the surfaces tested for smoothness with a sickle probe and then polished with pumice in a rubber cup. To keep the scalers and curettes perfectly sharp, small mounted arkansas stones used in the handpiece are necessary as well as the ordinary flat stone. As usually performed, scaling consists of removing supra-gingival calculus from the lingual surfaces of lower incisor teeth and then tobacco and other stains are polished off with brushes and pumice. This may be a beauty treatment but it is far less than the tissues deserve. The meticulous removal of every particle of subgingival calculus is one of the most difficult and exacting tasks that a dentist is called on to perform, but it is also one of the most valuable services that he can render his patients.

Food impaction is a problem in many mouths because it not only irritates the gingivae and the periodontal membrane but also causes teeth to move into positions where they are in traumatic occlusion. Some cases can be treated by replacing defective restorations, some require bridges or partial dentures to prevent drifting or extrusion and many cases can be treated by grinding plunger cusps and by contouring restorations to emphasise the marginal ridges. The gingivae may also be irritated by careless use of matrices, separators, rubber-dam clamps and ligatures or by restorations which have an excess or a deficiency of filling material at the gingival margin of a cavity. Damage to the tissues by faulty operating technique is particularly likely to occur when the tissues are anaesthetised. Before packing amalgam, the matrix band should be tested and a wedge used if necessary. As soon as the matrix band has been removed, carve and smooth the proximal portion of the restoration along its gingival border. This is done with amalgam knives, Ward's wax carvers or McCall's right and left posterior sickle scalers, Nos. 11 and 12, and should be completed before working on the occlusal. All restorations which are deficient at the gingival and many which have an excess or overhang will need to be replaced, but some of the latter can be corrected by the use of heavy scalers, flame-shaped finishing burs and Bunting's files.

Besides taking bite-wing radiographs for the information obtained about early proximal caries, the condition of the crest of the bone and the condition of old restorations, it is very good practice to take further bite-wing radiographs after treatment has been completed.

The last local irritation which will be considered is mouth breathing. Prolonged exposure to the air produces characteristic inflammatory changes in the gingivae because of the drying effect. The inflammation is confined to the anterior part of the mouth and may be seen on the labial and palatal in the upper jaw and on the labial in the lower jaw. At first the gingivae are red, oedematous and bleed easily but later there is a gradual transition to fibrous hyperplasia of the gingivae. This is often very marked about the onset of puberty. Treatment is divided into three parts:

1. Treatment of the nasal obstruction by the ear, nose and throat specialist.
2. Treatment to break the habit of mouth breathing.

3. Treatment of the effects of mouth breathing.

The last two are the responsibility of the dentist. In many cases if the habit has been established for some time, removal of the nasal obstruction is not enough and something positive has to be done to stop mouth breathing at night. The patient should be instructed to do deep-breathing exercises three times daily and before retiring at night and should be taught exercises to increase the tone of the orbicularis oris muscle. A mouth screen is the most positive method of ensuring nasal breathing — it can be made of acrylic, swaged aluminium or soft plastic sheet. For the young child or apprehensive patient who is frightened of a mouth screen, a piece of cellulose tape can be placed vertically to hold the edges of the lips in contact. The child can still talk and can breath through the mouth but a conscious effort is necessary. The screen or tape should be worn every night for from four to six weeks and then left out for one night so that the parent can check on whether the mouth is open or closed. If there is still any mouth-breathing tendency, it will be necessary to continue with the screen or tape for another week or two. The patient should be advised to use their mouth screen or tape for a few days after recovering from a "cold."

Treatment of the effects of mouth breathing will depend on the tissue changes that have occurred. If there is marked hyperplasia of fibrous tissue, removal of the cause will not be followed by a return to normal and it will probably be necessary to perform a gingivectomy to eliminate the false pockets, to leave a thin gingival margin and to produce deflecting contours interproximally. If the gingival enlargement associated with the mouth breathing is mainly due to inflammatory oedema, removal of the cause and gingival massage will allow the tissues to revert to normal.

When *all* the subgingival calculus has been removed it is necessary to instruct the patient in home care of the mouth.

Because of the lack of stimulation associated with a civilised diet, it is necessary for patients to clean and massage the gingivae each day to maintain optimum health. Unfortunately, this is not easy; it requires the sustained co-operation of the patient and the patient has to be taught by his dentist to use the toothbrush (and sometimes other aids) so that it will perform all its functions. However, this is the most useful service any dentist can render his patients and it is the duty of every dentist to make sure that he is qualified to give specific instructions appropriate to the individual needs of patients. When used in the proper manner, the toothbrush has the following effects:—

1. By alternatively applying and relaxing pressure and by frictional stimulation, the circulation of the blood is increased, thus bringing oxygen to the tissues and removing the products of cell metabolism and accumulation of inflammatory cells and tissue fluids. Because of anastomoses between the blood vessels supplying the gingivae, the alveolar bone and the periodontal membrane, improvement in the circulation in the gingivae also has a beneficial effect on the alveolar bone and the periodontal membrane.

2. The friction associated with the use of the brush keratinises the epithelium and removes food and other debris from the teeth and the gingivae.

Patients should be advised to buy two or three brushes so coloured or numbered that they can be used alternatively. The brush should have a small head with two or three rows of tufts with six tufts to a row. All tufts should be of the same height and, if filled with nylon bristles, the cut ends of the bristles should be polished. Patients will always ask "What toothpaste do you recommend?" and seem disappointed if told that no one dentifrice is better than all the others and that there is no scientific evidence, as yet, that any reduction in caries associated with the use of a particular dentifrice is due to anything more than the cleaning action of the toothbrush.

At least two appointments are necessary to teach a patient how to care for his mouth. At the first, explain why it is necessary to use a toothbrush and also explain that it is a massaging as well as a cleaning device. Having previously advised the patient about the type of brush to be used, select the method of toothbrushing best suited to the age, intelligence and interest of the patient and demonstrate this method first on a model and then in the patient's mouth. Have the patient use a hand mirror, first to watch your demonstration in his mouth and then to assist him to practise the technique himself. The patient should be told to bring a toothbrush with him at the next appointment because he will be expected to show whether he has mastered the new method. At the next appointment examine the mouth for areas that are being missed or for soft tissue abrasion. If the patient is missing certain parts, apply an iodine disclosing solution before drawing the patient's attention to them. Have the patient repeat the various steps in the technique that you have taught him so that you can correct any faults or omissions.

Of the many methods of toothbrushing that have been devised, the modified Stillman method is probably the best method for the average adult. The Charter's method is indicated where large interdental spaces are found either as a result of a gingivectomy, of advancing age or of attacks of acute ulcerative gingivitis.

Fone's circular method is a good method for an adolescent or for an adult who is not intelligent enough nor sufficiently interested to master the Stillman or Charter's methods. If used by adults, it usually causes some tissue destruction in the premolar and molar regions. Because the ends and not the sides of the bristles are used in this method, the bristles should be polished and should not be too stiff. The new multi-tufted brushes are suitable for this method although they are not much use for the Stillman or Charter's methods.

Other simpler methods of brushing are adequate for young children, but a horizontal "scrubbing" action is the least effective and the most harmful.

Of the various aids to oral hygiene, dental floss will clean proximal surfaces of the teeth but has no massaging effect on the soft tissues and can easily damage the papillae if used carelessly. Toothpicks, and interdental stimulators assist in cleaning but their main use is to massage the interproximal soft tissues. In doing this, they supplement the toothbrush in the region where it is least efficient. Interdental stimulators are preferable to toothpicks because they can be used anywhere in the mouth and at the correct angulation to produce deflecting contours interproximally. Mouthwashes containing any active drug should not be used continuously but a hot saline mouthwash dilates the superficial vessels and assists in the removal of debris which has been dislodged by the brush.

The dentist's third responsibility in the prevention of periodontal disease can be discussed under the heading of abnormal function.

Functional stimulation is necessary if the tissues are to remain healthy and therefore patients should be encouraged to use foods which require vigorous mastication and to have missing teeth replaced by bridges or partial dentures. Besides bringing teeth in the opposing jaw back into function, they prevent drifting and tipping of the adjacent teeth and so help to prevent food impaction, interproximal caries and occlusal trauma.

The commonest and most damaging type of abnormal function is known as traumatic occlusion or occlusal trauma. It can be defined as increased functional stress which is beyond the recuperative and adaptive capacity of the cells of the supporting tissues. The limits or the threshold of adaptive capacity are influenced by all those factors which affect the internal and external environment of the cells. These are such things as nutritional influences, endocrine activity, debilitating diseases and local trauma.

Increased vertical stress is well tolerated because the stress is borne by all the principal fibres but an increased lateral stress is resisted by relatively small areas of the periodontal membrane and these are then traumatised and degenerative changes occur. Prevention of occlusal trauma depends therefore on the treatment of incipient malocclusions so that the cusps and inclined planes make sliding contacts during all movements of the mandible and on promoting or imitating natural wear so that lateral stresses are progressively reduced. The following methods of treatment can all be of benefit:

1. Orthodontic treatment (where feasible).
2. Extraction of rotated, malposed, extruded or impacted teeth.
3. Oral rehabilitation by onlays, bridges, and partial dentures.
4. Occlusal equilibration by grinding.

As with other methods, this last method has limitations, particularly where there is considerable overbite, but most cases can be improved and many can be treated very satisfactorily.

In adjusting the occlusion by grinding, try and simulate natural wear as it is seen on the teeth of primitive peoples. When apportioning the load to be carried by the different teeth, consider the size and shape of the roots, the ratio of clinical crown to clinical root and especially the structure and the amount of supporting bone. Unless there are definite indications for it, grinding should not reduce the vertical dimension. Most of the grinding is done on the upper teeth and on the lingual cusps of the lower posteriors and it should be spread over a few appointments to allow adaptive and defensive reactions in the periodontal membrane and the pulp. If a tooth is very mobile or tender, grind sufficiently to take it out of occlusion for a time and, if possible, support it against lateral stresses by some form of splint.

Always polish the ground surfaces each time the occlusion is adjusted so that the teeth slide freely over each other and feel comfortable to the patient. While examining the occlusion and the supporting tissues of the teeth, be on the look out for occlusal habits which may cause extensive damage if unchecked. Some of these habits are occupational (e.g., seamstress biting thread), but many are neurotic in origin (e.g., bruxism).

Detection of habits depends on close observation of the patient, study of the type of damage produced and questioning as to occupation, hobbies, etc.

To recapitulate, prevention of periodontal disease by the general practitioner depends mainly on the following:—

1. The dentist carrying out treatment procedures in all branches of dentistry so that he does not cause periodontal disease by creating local irritants or by upsetting the occlusion.
2. Individual education and instruction so that the patient can take care of his own mouth.
3. Early recognition of incipient periodontal disease while treatment is still a comparatively simple procedure. This is a challenge to all dentists — it involves changes in our thinking and practice of dentistry. We should start to change now.

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Recent Advances in the Knowledge of the Dental Hard Tissues with Special Reference to Enamel*

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A fuller appreciation of the special pathology of the teeth can only come from an understanding of the similarities and differences between calcified tissues in general. The fibrous protein framework of bone, cementum and dentin is of comparable structure. Upon hydrolysis this organic matter gives an amino acid composition comparable to gelatin. Under the electron microscope it has been shown that these protein fibres have a sub-microscopic striation characteristic of collagen. The fibrous protein framework of enamel, on the other hand, gives no such patterns under the electron microscope. Hydrolysates of the enamel framework examined by means of paper chromatography reveal an amino acid ratio of histidine, lysine and arginine in the proportion of 1:3:10, which is very close to the ratio characteristic for a eukeratin (Block and others[1], Losee and Hesse[2]). Information about the evolution of the teeth, the embryological origin of the enamel from epithelial cells, and recent histochemical findings (Wislocki & Sognnaes[3]), are also in harmony with the conclusion that the bulk of the organic framework of enamel is a keratin-like substance. In addition to the fibrous protein framework, it appears that there is a small amount of widely dispersed ground substance, a carbohydrate-protein complex which may act as a coating of the fibres and possibly serve as a bond between the inorganic matter.

Examination of the teeth by means of polarized light and x-ray diffraction indicates that the inorganic portion consists of highly oriented apatite crystals. Except for size, these crystals appear to be similar in all calcified tissues: bone, cementum, dentin and enamel. Chemically, these crystals contain mainly tricalcium phosphate with smaller amounts of magnesium phosphate and calcium carbonate. In addition, spectrochemical and other analyses have shown the presence of numerous trace elements. It is not known whether these are intermixed with the main components or attached to the crystal surfaces. In fact, only one of these trace elements, namely fluorine, has been subjected to any extensive research.

Bone, cementum, dentin and enamel thus share three major components: first, the inorganic crystals; second, a fibrous protein framework, and third, a glyco-protein ground substance. This type of information, if confirmed by other methods, would tend to bring the enamel closer into the family of hard tissues than formerly believed. Yet the absence of adjacent and intrinsic cells places the enamel of adult teeth in a unique position among hard tissues (Table I).

In view of what has been said about the structure and chemical components, it is next of interest to consider the metabolic potentialities of the dental hard tissues. It is well known that the development and growth of the organic framework of the teeth are influenced by a variety of metabolic factors. This is clearly indicated by the sensitive performance of the odontogenic cells in deficiency states (Howe and others[4, 5]). Secondly, it is quite evident that the calcification of the organic matter of the teeth is just as much under the influence of various metabolic factors as are other hard tissues. That is well illustrated by

*Read at the 13th Australian Dental Congress, Brisbane, June, 1953.

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TABLE I

THE UNIQUENESS OF FUNCTIONING ENAMEL AMONG THE HARD TISSUES

MAIN COMPONENTS	BONE	CEMENTUM	DENTIN	ENAMEL
Inorganic crystals	Calcium phosphate-Apatite	Calcium phosphate-Apatite	Calcium phosphate-Apatite	Calcium phosphate-Apatite
Organic ground substance	Polysaccharide-Protein complex	Polysaccharide-Protein complex	Polysaccharide-Protein complex	Polysaccharide-Protein complex
Fibrous organic framework	Collagen	Collagen	Collagen	Keratin
Internal cells	Osteocytes	Cementocytes	Cell processes	Absent
Adjacent cells	Osteoblasts Osteoclasts	Cementoblasts	Odontoblasts	Absent
Blood vessels	Present	Absent	Absent	Absent
Adjacent fluid	Connective tissue fluid	Connective tissue fluid	Connective tissue fluid	Saliva

the dental damage caused by failures in the calcifying property of the diet during tooth development (Mellanby[6]). But once the tooth is formed and calcified and ready to erupt there is limited knowledge of further changes taking place in the process of maturation and function. An erupting tooth looks very much like one that is in function when examined with the eye and to the x-ray, as we use it clinically. However, certain areas of the most recently calcified enamel are more stainable than the more mature parts. Under the electron microscope enamel of erupting teeth does not appear fully calcified because the interprismatic region has more of a homogeneous type of organic framework characteristic of immature enamel. Furthermore, the studies with radioactive tracers have shown that the enamel, during and shortly after eruption, is far more capable of taking up radioactive tracers such as radioactive phosphorus, than is enamel that has been in the mouth for some time (Sognnaes & Shaw[7]). These are, then, a few clues to what may be important processes during and shortly following eruption of the teeth into the environment of the mouth.

What physiological changes is the enamel capable of undergoing after the teeth are in function? As a result of radioactive tracer work, the concept of turnover or exchange has been introduced: a give and take process on the molecular level, beyond the visualizing power of optical instruments. In experiments with bone and teeth it is becoming evident that these tissues are subject to such a process of turnover. So far, the subject has mainly been studied in regard to the inorganic fraction. On the surface of the crystals it appears as though such ions as phosphorus and calcium can be lifted off and replaced continuously (Falkenheim and others[8, 9]).

In order to elucidate this problem in living animals we have set up experiments as shown

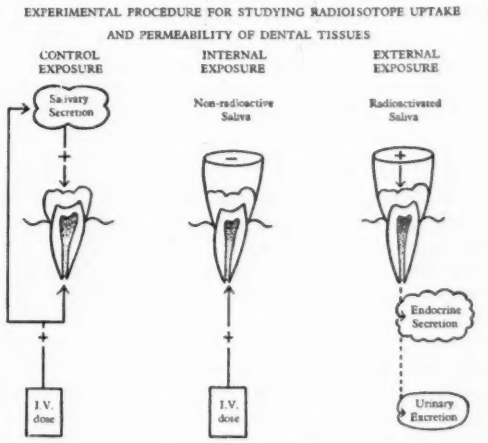

Fig. 1.—Procedures for radiotracer studies in Rhesus monkeys.

in figure 1. These procedures allow us to compare the radioisotope uptake from internal and external environment. Figure 2 shows two gradients in the distribution of radiophosphorus following intravenous P^{32} administration to rhesus monkeys. We have found that the radioisotope uptake in the dentin can be nearly abolished by removing the pulpal blood supply. The uptake in the enamel can be similarly abolished by preventing salivary access to the tooth surface. Comparison of several isotopes has provided certain clues to the relative importance of exchange, uptake and diffusion (Fig. 3).

With the little work done to date, comparing such tracers as radioactive phosphorus, radioactive iodine and tritium water, it is quite clear that various ions can readily penetrate through the dentin and enamel from the inside into the surrounding environment and from the external environment, saliva, through the enamel and into the pulp and blood stream

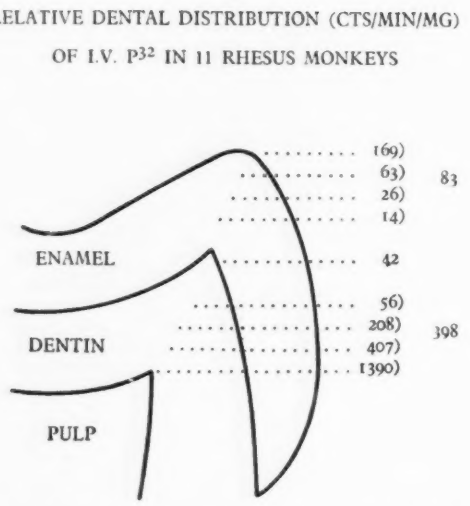

Fig. 2.—Radiophosphorus gradients in enamel and dentin following intravenous administration (from Sognnaes & Shaw, J.A.D.A., May, 1952).

(Sognnaes & Shaw[7]). This is not only true of phosphorus which is an integral part of the tooth and which may exchange with the phosphates on the surface of the crystals. It is also true of other elements, for example, iodine, which does not normally belong in the teeth but nevertheless passes through in both directions. Considering all the types of elements we take into our mouths, there is good possibility of ions passing through the dental hard tissues that do not belong there and, hence, may do more harm than good. Fortunately there is also the possibility that, once we learn more, substances may be introduced which may be useful to the maintenance of health in these tissues.

Being acellular and avascular it is evident that the enamel is completely devoid of any capacity of repairing itself in the ordinary biological sense of the word. But regardless of the biological aspects, the possibility does exist that the salivary and pulpal environment may effect the crystal growth, crystal poisoning and crystal repair on a molecular level.

The behaviour of the organic part of the enamel remains to be explored. Is it static and irreplaceable once laid down? Such a casualty as a crack in the enamel, which is almost unavoidable, often appears to be filled in with organic matter. It is not known whether this originates from the saliva or the pulp; but one wonders as to whether this organic matter, once in, may become a nucleus for secondary deposits of inorganic matter and, hence, serve as a crude form of repair.

For many years the enamel was considered an inert shell of lime salts, a passive prey to acid formed by bacterial fermentation of carbohydrates in the oral environment. This, then, was a fundamental conclusion upon which much of subsequent caries research was to be based. Over the years it has become increasingly apparent that Miller's[10] concept of enamel and, hence, of caries, was in part derived from artefacts, resulting from technical difficulties, rather than from biological peculiarities unique to the enamel.

Progress of our knowledge along these lines is closely linked to recent progress in tools of research. Stimulated by the work of Bodecker[11] who almost alone kept the issue alive for many years, there has been during recent years an increasing interest in the organic components of the enamel. As a result it has been revealed that, in addition to the inorganic crystals, the enamel is permeated by a delicate organic framework. What can be seen under the ordinary light microscope of carefully decalcified enamel is essentially a meshwork of organic matter surrounding each individual prism unit (Sognnaes 1949/50[12]).

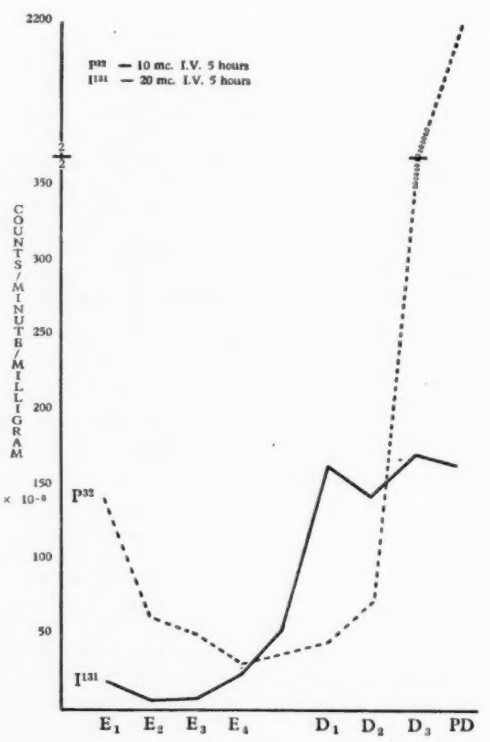

Fig. 3.—Comparison of radiophosphorus and radioiodine gradients in enamel and dentin.

This unit is about 5 microns in diameter and it takes about 10,000,000 of these prisms to build the enamel of a molar tooth. When the

minerals are completely removed the prism core looks empty under the ordinary microscope. The space between the prisms, where the organic matter is concentrated is only a micron or less in width. Hence it has proved impossible to obtain more detailed information with the ordinary light microscope.

Since then the electron microscope has revealed more minute organic components which were not previously thought to exist (Scott and others[13] and Sognnaes and others[14]). In the main this new tool has revealed that the core of the prism is not wholly inorganic but that it is permeated by numerous delicate organic fibrils which surround what appear to be individual inorganic crystals. Between the prism sheaths themselves can likewise be revealed many minute crystals, each surrounded by this fibrillar organic framework as shown in figure 4. Considering the dimensions of these organic building blocks, it is quite evident that the dimensions of the oral microorganisms are so great by comparison that both the organic and inorganic matter—or the bond between the two—have to be deficient or removed before any organisms can enter the enamel.

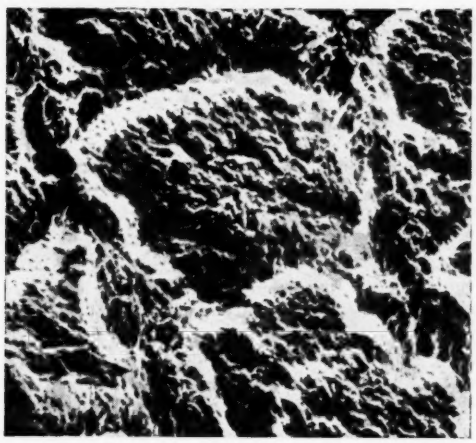

Fig. 4.—Electron microscopic structure of enamel (x 20,000) showing submicroscopic fibrillar matter within and between the prisms (From Scott, Ussing, Sognnaes & Wyckoff, J. Dent. Res., Feb., 1952).

A more fundamental question has at the same time been forced upon us. The inorganic enamel crystals that build the prisms are apparently not—as previously thought—stacked up against each other as so many pencils, one crystal hanging on to the next. In view of what has been shown in the high power electron microscope, it appears instead as if every inorganic building block is holding hands, as it were, by way of the organic matter in between. What is the nature of this bond that is holding so firmly together all the hard tissues of the body? Normally the crystals do not fall apart. On the contrary, the combined inorganic and organic units are far stronger than is each separately. Hence the bond between them must serve a very important function. The nature and mechanism of this bond is of paramount interest from the standpoint of tooth development and caries. At the moment we are in the dark on this point. To answer this question, we need more information about the chemical nature of the organic components which are interspersed between the inorganic crystals.

It is thus apparent that much of our efforts must still be directed towards further studies on the structure of the tissues involved in dental disease with respect to morphology, chemistry and physiology. Such information should help to set the stage for further exploration of the incipient pathological changes associated with dental caries and, eventually, enable us to interfere intelligently with those changes before the irreversible carious destruction sets in.

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Fibrous Dysplasia of Bone*

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The term fibrous dysplasia has been applied generally throughout the years to a wide variety of conditions, and I must make clear to what pathological state I refer in this paper. Perhaps the best way to do this will be to trace the process by which this specific entity has been separated from the heterogeneous group of diseases originally described as fibrocystic disease of bone or, perhaps just as commonly, as osteitis fibrosa.

In 1891, Von Recklinghausen gave the first recognized accurate description of a disease characterized by generalized fibrosis and cyst formation of bone. This type of lesion then became known as generalized osteitis fibrosa or Von Recklinghausen's disease of bone. Over the years many others spoke of it under other names, emphasizing aspects of the disease which some cases showed in a gross form; such names as multiple giant cell tumours, fibrocystic disease of bone, osteodystrophia fibrosa, and osteomalacia with multiple giant cell sarcoma. A connection of the function of the hyperplastic parathyroid with malacic bone disease in general was suggested early in the century. Enlargement usually of one but sometimes of more parathyroids had been found in porotic diseases of bone generally and, in particular, in "osteitis fibrosa cystica." Thannhauser[1] lists eleven reports of this up to 1925. These discoveries were substantiated by Collip's isolation of parahormone and by other studies indicating that the parathyroids were associated with the physiological regulation of calcium metabolism. It only needed Mandl's report of the almost miraculous recovery of a patient after the successful extirpation of a parathyroid adenoma, in a case of osteitis fibrosa cystica, for Recklinghausen's disease of bone to become a pathological entity, identical with hyperparathyroidism and specific as regards both its etiology and treatment.

It soon became evident, however, that there were some cases showing apparently typical lesions of hyperparathyroidism which did not fit into this classification. In surveying the cases of hyperparathyroidism reported in the literature, two theories are found:—

(1) That only those fibrocystic bone changes which display simultaneously high serum calcium and evidence of a negative calcium balance are a result of hyperparathyroidism, and

(2) That hyperparathyroidism is the only etiological factor in generalized osteitis fibrosa.

This second theory, that hyperparathyroidism is found in all cases of osteitis fibrosa cystica and is considered to be the etiology of this condition, does not conform to the facts observed in clinical and post-mortem examinations. Approximately 50 per cent. of all cases diagnosed as generalized osteitis fibrosa displayed normal serum calcium values, and normal calcium balance; and no histological signs (e.g. agglomeration of osteoclasts) were found. Moreover, even in those cases with symptoms of calcium imbalance treated with parathyroidectomy, the fibrocystic bone changes did not disappear in every case. Perhaps even more damning were those, mistakenly diagnosed as osteitis fibrosa associated with hyperparathyroidism, in which no evidence of parathyroid hyperplasia was evident on surgical exploration. Mallory[2] mentions such a case in which removal of two normal glands was followed by unmistakable signs of tetany.

Albright and others[3] have reclassified these aberrant cases of generalized osteitis fibrosa into a separate clinical entity. For not only are these characterized by normal calcium values, associated with fibrocystic bone changes, but they also show a unilateral involvement of the skeleton, pigmentation of the skin and various endocrine disorders, all of these symptoms varying in degree from patient to patient. To this disease they give the name osteitis fibrosa disseminata, and osteitis fibrosa generalizata (or Von Recklinghausen's disease of bone) is reserved for the hormonal disorder of hyperparathyroidism with its associated osseous lesions. In this respect, it has been said that a number of the original cases upon which Von Recklinghausen's Festschrift paper was based were actually cases of osteitis fibrosa disseminata[4].

Working independently, Lichtenstein had investigated this disease and in 1938[5] published his findings designating this same disease, osteitis fibrosa disseminata, as fibrous dysplasia of bone—a name derived from his conception of its pathogenesis, as a result of perverted activity of tissue formation.

This, then—be it known as fibrous dysplasia, Albright's syndrome, osteitis disseminata, or any one of the thirty-three other titles from which Lichtenstein and Jaffe have collected relevant cases—is that disease which I wish to discuss today, that is to say:

> A condition affecting one, several or many bones, the graver cases of which may present abnormal pigmentation of skin, premature sexual development, hyperthyroidism and still other extra-skeletal abnormalities[6].

*Read at the 13th Australian Dental Congress, Brisbane, June, 1953.

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Although no pathologist would take such a specialized view as to refuse to study anything which appertained only to his particular sphere of inquiry, yet such an imposing array of general symptoms as have been presented in the literature on this subject might cause you to wonder why this paper has been presented at a Dental Congress. What I wish to emphasize is that the severity of this disease varies greatly and, although cases have often been reported showing the triad of skeletal abnormalities, skin pigmentation and precocious puberty, far oftener the disease is represented by localized fibrotic changes, frequently limited to one bone. These different forms of the same disease have been designated polyostotic and monostotic respectively.

That these polyostotic and monostotic types are variants of the one condition was suggested by Lichtenstein and Jaffe in 1942[6], and later authors have agreed with this. I quote:

> The cases we have seen do not include many in which the skeletal involvement was dramatically severe, although such cases are rather heavily represented among the published instances of the disease. On the other hand, we have seen relatively many of the cases in which the disease was of a milder type . . . These cases, and especially those showing involvement of one bone only, are, we believe, underrepresented in the literature, being often interpreted as instances of "localized osteitis fibrosa" and therefore remaining unreported as not being of special interest. The skeletal lesions, however variable their extent and severity, appear to be analogous in all the cases, thus constituting the common factor of the disease.

We are, of course, primarily interested in this monostotic type, represented by localized bone lesions and the absence of any of the extra-skeletal abnormalities of skin pigmentation, precocious sexual maturation in the female, hyperthyroidism, or any of the other developmental abnormalities which may be present in the more florid expressions of the disease.

Clinical manifestations.

The clinical manifestations of this disease in those cases represented solely by lesions in a jawbone are generally slight. The patient may give a history of a long-standing, very slowly expanding swelling. A swelling of the affected maxilla or mandible is frequently noticed by the time the patient has reached adolescence. Infrequently do the cases come to clinical light after the age of 30-35 years. The swelling usually first draws attention to the condition, for seldom is the lesion itself painful. Occasionally, the facial asymmetry is so slight that it has gone unnoticed by the patient until a localised infection in the region led the patient to seek dental care, this, in turn, leading to the discovery of the abnormality in the jaw.

In only occasional cases is there any history of rapid enlargement of the affected bone and any pain or discomfort is produced only by an intercurrent infection, or is referable to pressure from the expansive growth. There is no tenderness to palpation or any marked disturbance of function.

Radiographically, affected bones, if the lesions are of long standing, will show some expansion of their contour. The cortex is thinned from internal resorption and is usually still recognizable as the cortex, there being little periosteal reaction to the expanding lesion unless it is associated with trauma and infection, or pathological fracture. In the maxilla, the growth may extend into the antrum, occasionally to the extent of almost obliterating that cavity.

The main body of the lesion presents many varied appearances. It may be a circumscribed area of radio-opacity, strongly resembling a cystic condition, though it contains, of course, a mass of fibrous tissue. Depending upon the degree of metaplastic ossification taking place throughout the lesion, the radiographic picture will vary through all grades of density from the totally rarefied lesion, through lesions showing a mottled and stippled appearance to completely dense, sclerotic overgrowths. In some, the stippling is often due to the presence of islands of ossifying cartilage. Any of these lesions may be single or multiple and localized to one jaw or one side of the mouth. In rarer cases they may be more widely distributed.

The character of the replacement tissue in these skeletal lesions is fibro-osseous. As is readily evident from a radiographic survey, there may be considerable variation in the character of this tissue from lesion to lesion and even within different regions of the same lesion, particularly in regard to the proportion of osseous to fibrous elements. The tissue has been described as tough, compressible, and evenly whitish or reddishly speckled. It may be gritty throughout from the presence everywhere within it of newly formed trabeculae of immature bone, the characteristic replacement tissue of fibrous dysplasia. Areas with few metaplastic trabeculae are found, and in other regions, common in mature lesions, the tissue is densely ossified with little fibrous tissue often reminiscent of an eburnated osteoma.

In accordance with Lichtenstein and Jaffe's concept of the nature of these lesions, the essential microscopic component is fibrous connective tissue. Corresponding to the microscopic features of the lesion, this fibrous tissue varies from place to place in its detailed histological composition. Apart from the osseous and cartilaginous elements which it may contain and which represent metaplastic changes,

this connective tissue in some places or throughout a lesion may be rather cellular. In such cases it is composed of immature, small, slender, spindle cells in rather loose and whorled arrangement. On the other hand, it may be poorly cellular, and highly collagenous. Lichtenstein and Jaffe suggest that this highly collagenous tissue together with oedematous, myxomatous and cystic types represents modifications of the basic tissue—the rather loosely whorled and spindly connective tissue.

The connective tissue on the whole tends to be relatively avascular, though sporadic fields occur dominated by numerous enlarged and engorged, thin-walled blood vessels. Surrounding these, blood extravasations are common, such fields showing up in the cross section as reddish zones. Groups of multinuclear cells may be prominent in relation to such areas. These are not similar to those of giant-cell tumour and they represent a phagocytic reaction to the blood extravasations. A few small lymphocytes or other mononuclear cells may occur within the stroma of the lesion, their presence being of little significance.

Trabeculae of bone formed through metaplasia are common. Corresponding macroscopic evidence is the characteristic grittiness of even apparently soft fibrous tissue, with its radiolucent appearance. On the other hand, the dense, sclerotic overgrowths are formed by the same process. These isolated trabeculae and dense masses of metaplastic fibre bone are irregularly dispersed within the fibrous tissue, not following any regular pattern, and they themselves are of variable size and contour. Osteoid tissue may occur and occasionally small islands of hyaline cartilage are present embedded in the connective tissue. They are generally less than 1 cm. in diameter and tend to become calcified peripherally and undergo some degree of ossification.

Cyst formation, once described in association with most fibrodysplastic bone lesions as "fibrocystic disease of bone," is not common and such a term is not justified.

It will be agreed, I think, that fibrous dysplasia presents as a distinct picture. Its pathology as a whole is of a fixed pattern and the individual lesions are distinctly limited both in extent and, more importantly, in histological structure. Indeed Lichtenstein and Jaffe[6] can state:

> . . . a single glimpse of the character of the dysplastic connective tissue and of the primitive new bone within it, even in the absence of cartilage foci, will very often suffice to indicate the anatomic diagnosis.

Even though the form of the lesions may vary from loose connective tissue to dense, sclerotic, bony overgrowths, the basic pattern might easily be conceived as similar in all the lesions. The different histological features, then, are merely varying components of a single process.

Opinions will differ, however, as to the specificity of these lesions, and they have differed quite markedly to date. Indeed, although this group of tissue changes with their clinical manifestations, which has been described, is definitely limited in extent, it is by no means representative of a single disease process as present day oncological classification stands. From the description I have given, fibrous dysplasia can very easily be classified as "ossifying fibroma," "fibrous osteoma," "osteoma," or "osteofibroma," depending upon one's opinion of the nature of the process.

Present day interest in this subject is centred about this discussion of the nature of these lesions. Over the last 7-8 years it has been the main feature of articles written about this group of diseases (Schlumberger[7], Mammel[8], Crane and Wolgamot[9], Scarff and Walker[10], Rushton[11], Thoma[12], Jaffe[13], Berger and Jaffe[14] and Smith and Zaveleta[15].

Lichtenstein in 1938[5] and Lichtenstein and Jaffe in 1942[6] conceived of fibrous dysplasia, in both its polyostotic and monostotic forms, as a congenital anomaly of development.

> As to the skeletal lesions in particular, these apparently result from perverted activity of the specific bone-forming mesenchyme, producing a focus or foci of involvement in one bone, a few bones, or many bones. In a sense, the individual bone lesion may be conceived as a hamartoma — i.e., a tumor-like malformation resulting from flaws of development and characterized by defects of tissue combination. We have tried to suggest this interpretation of the derivation of the bone lesions by the name fibrous dysplasia of bone.

It will be seen that fibrous dysplasia is thought to involve no neoplastic change, being more of the nature of a developmental hyperplasia.

On the other hand, other lesions, such as ossifying fibroma, scarcely able to be differentiated from those of fibrous dysplasia, are classified as benign neoplasms, though extensively differentiated and of a low potential. In spite of this there is almost complete agreement that cosmetic operations are all the treatment that is required, though occasionally further enlargement after such an operation will take place.

Jaffe in 1938[6] considered fibrous osteoma of the jawbones was closely analogous pathologically to the condition which, in relation to limb and trunk bones, was being designated as fibrous dysplasia. With subsequent experience both with fibro-osseous jaw lesions and with fibrous dysplasia, he maintained his previously held ideas. It appears, then, that since there is a lack of knowledge of any generalized affections associated with these

lesions, the possible identity of fibrous dysplasia and ossifying fibroma must be sought in the histological appearance of the two lesions. And Schlumberger in 1946[7] can follow such an investigation with:

> Since, therefore, histologic criteria *do not* exist by which fibrous dysplasia of the maxilla or mandible can be distinguished from so-called ossifying fibroma of these bones, and because their clinical behaviour is identical, it is concluded that the ossifying fibroma is a variant of monostotic fibrous dysplasia.

On comparison, the lesions of fibrous dysplasia and those of ossifying fibroma (or fibro-osteoma) are remarkably similar. Thoma [12, 16] describes these lesions as benign tumours of osteogenic cells originating in the marrow spaces of the spongiosa. Since these same osteogenic cells may show various degrees of differentiation in individual tumours, producing a variety of intercellular substances which may become partly or completely calcified, various types of fibro-osteomas have been described. These have been classified on this basis as—

(1) ossifying fibroma;
(2) fibro-osteoid osteoma;
(3) mature fibro-osteoma, including the dense sclerosing type.

These are described as follows:

(1) *Ossifying fibroma.* The term, ossifying fibroma, has been suggested by Phemister and Grimson. The fibrous tissue is overabundant though bone trabeculae may be beginning to form. The ossifying fibroma, therefore, may be the primary stage of the fibro-osteoma. It is made up occasionally of very immature connective tissue cells, giving the impression of a fibrosarcoma, though no mitotic figures may be present.

(2) *Fibro-osteoid osteoma.* In this second type, the osteogenic connective tissue cells fill the marrow spaces, forming immature, so called fibrillar, woven bone from condensed, hyalinized, intercellular substance, which is deposited around them, while the trabeculae of old bone are undergoing osteoclastic resorption. In other instances, the bone is partly or completely replaced by trabeculae of osteoid. The fibrillar bone is formed by fibroblasts of a low level, while the trabeculae are produced by fibroblasts of a higher level of differentiation. The newly formed bone has irregularly placed osteophytes and there is evidence of apposition and resorption.

(3) *Mature fibro-osteoma.* In a third type more mature bone is formed. The new bone trabeculae now become completely calcified, but the marrow remains cellular and fibrous in nature. In older individuals, we may see the sclerotic type. The marrow becomes acellular, the bone is well calcified and shows curvilinear markings. These are due to broad cement lines which stain deep blue and provide a mosaic pattern.

If, as Lichtenstein and Jaffe claim, the histological picture is sufficiently distinctive to be regarded as specific, and even pathognomic, then these lesions described as fibro-osteomas must surely be classified with fibrous dysplasia, as Jaffe has lately maintained. Certainly, granted the histological picture is the criterion, no worker claims that there is any significant difference between the lesions. In the same manner, those who claim that the disease represented by this lesion is neoplastic do so only on the basis that it is an expansive new formation.

The first paper proposing that these jaw lesions are of a neoplastic nature appeared in 1937[17]. This paper by Phemister and Grimson was important in that it attempted to present a unified conception of these jaw lesions on the basis of clinical, radiographic and pathologic findings. Up until that time, such lesions had been variously classified as fibro-osteoma or as localized osteitis fibrosa, osteodystrophia, intra-osseous epulis or hypertrophic localized osteitis. Neither Albright's nor Lichtenstein's papers had yet appeared.

It will be observed that most of this latter group of names suggests some sort of reparative process, commonly thought to be associated with that vaguely defined disease, "osteitis fibrosa." It seems to be on this account that Phemister and Grimson insisted on the term fibro-osteoma to distinguish this group of jaw lesions as essentially neoplastic and as a reparative process of a variety of local or systemic factors. The lesions certainly do not have any apparent relationship to injury and subsequent reparative processes; but to designate them neoplastic requires further knowledge of the process.

To a certain extent those today who consider these lesions as neoplastic still object to the conception of a developmental hyperplasia on the basis of Phemister and Grimson's reasonings. However, these objections were originally directed against the idea that the disease was a reparative reaction following injury. This idea of the connection of the disease with injury is not in any way part of Lichtenstein and Jaffe's conception of its nature and generally, to date, there has been no specific objection to these ideas of Lichtenstein and Jaffe and their followers, that is, of course, only so far as I have been able to discover from the literature on this subject.

For example, Thoma in 1950[12] is still classifying monostotic fibrous dysplasia with localized osteitis fibrosa and objecting to the classification of fibro-osteoma as such. He can, on the other hand, make these statements bearing positively on the nature of these jaw lesions:

> The fibro-osteoma is a tumour which causes the marrow spaces to be filled with actively proliferating young osteogenic connective tissue cells. As the

tumour tissue accumulates, resorption of the bone trabeculae takes place and later when more tissue is formed, the region of the jaw which contains the tumour expands. This process is similar to the development of a tumour formed from haemopoietic tissue . . .

Thoma continues:

Therefore I feel with Furedi (1935) and Phemister and Grimson (1937) that the fibro-osteoma is a tumour (i.e. a neoplasm) which arises in the spongiosa.

Conclusion.

If equal credence is given to the schools of thought on this subject, it is evident that there is no easy solution to the problem of the nature of these lesions, variously named "fibro-osteoma," "ossifying fibroma" or "fibrous dysplasia" and, at the same time, classified into separate etiological groups. The problem can be represented by two propositions.

Either (1) We are concerned with two apparently identical disease entities which represent two different pathological processes,

or (2) The two disease entities represent the same process, either neoplastic or not.

I think it is relevant here to make several comments on this.

1. The term "hamartoma" originally used by Albrecht, I believe about 50 years ago, has, with the work of Willis and Lichtenstein and Jaffe, been revived in pathological literature. These hamartomas have been described as tumour-like malformations resulting from flaws of development and characterized by defects of tissue combination. They are thus strictly developmental malformations, though this does not necessarily imply that they are congenitally acquired. Malformations, generally, even those involving an excess of tissue, such as accessory spleens, supernumerary digits, etc., would not be mistaken for tumours. Confusion has occurred most frequently as regards those malformations in which the various tissues of the part are present in incorrect proportions or distribution, with prominent excess of one particular tissue — these are properly designated as hamartomas.

Their behaviour seems to be known quite satisfactorily for the clinical pathologist to study them, as is evidenced by the treatment of fibrous dysplasia, but the more important study of the nature of these "congenital" errors of development has been neglected — and there I am probably exceeding the bounds of our knowledge.

What is the nature of a hamartoma? And I suppose an equally pertinent question would be to ask—"what is the nature of the neoplastic process?"

2. Just where does the boundary lie between a hamartoma and a neoplastic lesion? I fear that, in contrasting the two theories of the nature of the jaw lesions under discussion, I have presented them as mutually exclusive classifications. Might not it be otherwise?—that these apparently identical lesions represent a borderline condition where the developmental error, as represented by the malformation, approaches that fault, developmental or not, which results in a potentiality for unlimited growth, the neoplasm.

By this I do not intend to classify these lesions into yet another category. Rather do I want to point out that neither the neoplastic state nor the normal state of tissues has any definite boundaries. Granted, then, that there is some degree of truth implicit in our pathological diagnosis implied by classification, may not this lesion represent a condition about which we should ask, not "Is this lesion neoplastic or not?", but "What neoplastic potential has this lesion?"[18]

This is the possibility which presents itself logically to the author from a survey of the literature on the subject.

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A Plea to Save Lower First Permanent Molars in Children*

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Much has been written about the harmful effects of premature loss of deciduous teeth; little has been said of the undesirable sequelae of loss of first permanent molars. The purpose of this paper is to show that these undesirable features are far more noticeable if the mandibular first permanent molars are lost in the important period of the mixed dentition.

Loss of these molars has serious results for the child. It leads to tooth tilting and drifting, periodontal pockets, incisal overbite, and an artificially produced distocclusion. Every effort should be made to save these lower molars, for their loss ruins many mouths and often prejudices an ideal result if orthodontic treatment is instituted.

Our Dental Education Is Not "Getting Across."

The mandibular first molars are usually the first permanent teeth to appear in the mouth. Normally they erupt behind the deciduous teeth with no displacement, and often come into the mouth unknown to child or parents. Because of this they are frequently mistaken for deciduous teeth, and so they are quite often allowed to decay because the parents place little store on deciduous teeth and expect them to "fall out" in two or three years' time. In a large number of such cases the dentist does not see the child until it has become too late to save these teeth permanently.

The great majority of Australian children allow their teeth to decay because they have never been shown why they should keep their gums and teeth healthy and clean, nor how or when to clean them. Our profession must accept much of the blame for this failure. Our dental health education should commence by teaching every child and parent that the food must be removed from the teeth and gums immediately after each time of eating, and asking the parents to see that this is carried out effectively. It is believed that proper oral hygiene can reduce the incidence of new dental caries up to 50-60%[1].

Other Preventive Measures.

The task of denture guidance can be a relatively simple matter if the following preventive measures are taken to reduce the incidence of caries:

i. Early diagnosis by the aid of bitewing radiographs.

ii. Application of silver nitrate and sodium fluoride where practical.

iii. Correct and balanced diet.

iv. Prompt and careful restorations.

Radiographs are a basic diagnostic necessity. Routine bitewing pictures taken every six months for children (or even nine months) will show up nearly all caries. It is then easily repaired, and allows all the deciduous teeth to be held until their normal time of shedding. Failure to use x-ray inspection is surely negligence.

We must also stress here the danger of a small undetected decay on the distal surface of the second deciduous molar. When these teeth are either extracted or shed they often have caries on the distal surface near the gingival margin, and we find caries on the mesial surface of the first permanent molar.

The Function of the First Permanent Molars.

From the standpoint of occlusion the important function of the first permanent molars is to establish and maintain the arch and jaw relationships in the three dimensions of space during the period of the mixed dentures. As Seward[2] has pointed out, the design of the human dentition is such that the mandibular teeth depend for their support much more on their line of contact points than on the fibrous attachment of the roots, the reverse being true for the maxillary teeth. In the mandible the continuity of the contact points ensures the stability of the arch; loss of a tooth breaks the continuity of the contact points and leads to results to be outlined below.

In deciding whether carious lower molars of a patient are to be preserved or extracted we must ask ourselves the question, "Can these teeth be expected to last thirty years?" If after careful inspection we are satisfied the answer is "yes," then the molars should be perfectly restored and thereafter maintained in excellent condition. If we come to the answer "no," they should be extracted according to the plan to be stated later. This is the appropriate place for an appeal for perfect restorations.

Results of Loss of First Permanent Molars.

Extraction of first permanent molars during childhood is followed by certain changes in the dentition, some desirable and some not.

Early extraction (that is, before about 9½ years). Extraction of the first permanent molars early results in:

(a) well shaped arches with regular teeth in over 80% of cases;

* Read at the 13th Australian Dental Congress, Brisbane, June, 1953.

(b) marked reduction of the incidence of interproximal caries, and virtual caries freedom for many years;

(c) no impaction of third molars.

For these reasons the method of extracting the four first permanent molars early is largely practised in England[3].

However, on the debit side,

(a) very often the mandibular teeth with their supporting bones do not develop their full distance anteriorly, with the result that a normal occlusion or a Class I malocclusion changes into an artificial distocclusion. The mandibular incisors, no longer opposed, continue to erupt into the palate, their alveolar bone accompanying them[4]. A deep incisal overbite is thus also produced.
To achieve normal occlusion both the above sequelae involve lengthy corrective treatment with fixed appliances on the teeth of both jaws, a costly procedure;

(b) the mandibular second permanent molars often tend to tip lingually in moving forward to close the first molar spaces;

(c) there is a tendency for rotation of mandibular teeth;

(d) the mandibular second premolars tend to occupy some of the space formerly taken up by the first permanent molars, especially if the mandibular second deciduous molars have been lost prematurely.

Late extraction. Extraction of first permanent molars after the eruption of the second molars leads to undesirable results in the mandibular arch but often has beneficial results for the maxillary arch, probably because of the more cancellous nature of maxillary bone.

(a) it is difficult to close the lower molar space even with fixed appliances; when the second molars are moved forward mechanically to the second premolars they often tend to tip lingually;

(b) if no mechanical measures are taken to close the lower first molar space, three things happen:
i. the second premolars tip distally;
ii. the second molars move forward for some distance at right angles to the occlusal plan; but at the same time
iii. the second molars tip mesially to a considerable degree.

(c) this mesiodistal tipping of teeth in an attempt to close the lower first molar space produces a severe periodontitis in many instances on the sides leaned to between the second premolar and the second molar.

(d) imbricated incisors do not naturally move into good alignment following late extraction of the first permanent molars; their correction at this age usually requires the employment of fixed appliances.

We should always bear in mind that since bone growth goes on to the age of 18-25 years, any changes in the development and growth of the jaws caused by the extraction of teeth will go on to this age. This is one of the reasons why relapses sometimes occur in treated malocclusion cases if the lower first permanent molars have been extracted. This can be seen in the post-retention period of treatment when the incisal overjet and overbite increases; what appears to be a lingual "collapse" of the lower incisors is really due to the fact that the forward growth of the mandibular arch lags behind that of the maxillary arch.

The question of whether the extraction of the four first permanent molars is the best method of preserving the dentition in its optimum state and the correct time to extract has by no means been finally decided. We have to weigh the pros and cons carefully and take every factor into consideration. Each case must be assessed individually on all the evidence. Then, in the final analysis if we are confronted with a choice of caries or malocclusion we must remember that deformity does not kill.

The Extraction of First Permanent Molars in Malocclusion Cases.

As we have seen, loss of first molar teeth in mouths with normal occlusion without subsequent control of the direction of movement of the remaining teeth usually leads to malocclusion. But extraction of first molar teeth in mouths showing malocclusion without control of the direction of movement of the remaining teeth nearly always leads to aggravation rather than improvement of the existing malocclusion.

Extraction from existing distocclusion. The loss of maxillary first permanent molars in distocclusion cases is not usually followed by self-correction because of the comparatively slight distal movement of the maxillary premolars and the relatively greater mesial movement of the maxillary second molars. The loss of the mandibular first permanent molars from an existing distocclusion usually increases the degree of malocclusion since the mandibular teeth with their supporting bones do not develop their full distance anteriorly. From the viewpoint of occlusion the early extraction of the lower six-year-old molars in a Class II Division I (Angle) malocclusion may turn it into a Class II Division II (Angle) malocclusion.

When the upper arch, teeth and musculature, is forward of the lower, normally or abnormally, there should be no loss of teeth from the mandible. It is interesting to recall that Angle said that the space should be retained with an appliance if the lower six-year-old molars have to be lost in such cases.

Extraction from existing mesiocclusion. The general principles given above for extraction from an existing neutrocclusion or an existing distocclusion do not apply for mesiocclusions (Class III, Angle), and no certain result can be expected in these malocclusions if the lower six-year-old molars are extracted because the tongue may hold the arch size in these cases. Loss of any maxillary teeth, and particularly that of the first permanent molars, in mesiocclusion cases often increases the degree of the malocclusion.

Some years ago Salzmann[5] analyzed the distribution of malocclusion (according to the Angle classification) in 500 adolescent patients after the extraction of first permanent molars, and compared this with the estimated distribution before the extraction (Table I). Now, although his work is not statistically valid, it shows an interesting trend.

TABLE I.

Angle Classification	After M1 Extraction	Before M1 Extraction (Estimated)
Class I	189 (38%)	365 (73%)
Class II	306 (61%)	130 (26%)
Class III	5 (1%)	5 (1%)

After the first molars are extracted, cases in Class I tend to change to Class II malocclusion, while both Class II and Class III cases tend to remain in the same classification as before extraction.

The Best Time to Extract Unsavable First Molars in Planned Treatment.

The main purpose of this paper is to show the importance of the first permanent molars, especially the lowers, and to appeal for their preservation if at all possible.

There are times, however, when for the benefit of the mouth as a whole our plan of treatment demands the eventual removal of the four six-year-old molars. In the present state of our knowledge, the best time to extract the lower first molars in such cases is at about the age of eleven when the unerupted lower second molars are just emerging from their bony crypts. The second molars ultimately move forward into the mouth with the least mesial tipping. Therefore, where lower first molars will eventually be lost because of caries or unsuccessful conservative work, every attempt including pulpotomy should be made to keep them in the arch until the lower premolars have erupted into occlusion with their upper antagonists. (Remember that often there is not enough room for the eruption of the four second premolars, and the molars may have to be extracted earlier.)

When the eruption of the lower second molars is so far advanced as to necessitate removal of the lower first molars before the full eruption of the premolars, most authorities teach that some provision should be made to maintain the normal vertical height of the jaws. This is usually done by fitting a plate with a small bite plane palatal to the upper incisors, upon which the lower incisors rest when the jaws are closed. The plate may be made and the vertical height adjusted before the teeth are extracted; after extraction it is placed in position so that the vertical height will be maintained until the complete eruption of the premolars. It is interesting to note in passing, however, that, if the freeway space is constant throughout life as Thompson says, the bite should not close when the molars are extracted and there should be no need for any appliance to maintain the vertical height.

In Class I and Class II cases of malocclusion where lower first molars have to be lost the plan of treatment should include extraction of maxillary teeth as well. For in both these types of malocclusion the malrelation of the teeth of the two jaws to their containing bones and musculature is frequently such that the upper teeth to be extracted should have an equal or greater number of cusps than the lower teeth to be extracted. Hence the teeth of choice for extraction in these cases are usually the maxillary first molars. If upper first molars are also to be lost their extraction is usually delayed until the upper second molars are well erupted in order to prevent rapid forward movement of the latter teeth.

Conclusions.

The first permanent molars are the most important teeth in the dental arches, and their loss usually has serious consequences for the dentition as a whole. Every device known to preventive dentistry should be employed to preserve them, and if they will have a normal dental life they should rarely be removed.

Loss of four first molars from a child with normal occlusion or a Class I malocclusion (by far the most commonly occurring malocclusion) usually results in an artificial distocclusion with deep incisal overbite, tooth leaning and drifting, and periodontitis. These results occur much less frequently if the correct premolars are extracted at the right time.

The essence of children's dentistry is planned treatment. We should always endeavour to have the patient's treatment

under our control alone, and not be forced into treatment which we know will have undesirable results. It is believed that the application of the principles given above will do much to improve the oral status of our child population, and pave the way for a healthier generation in the future.

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Factors of Skeletal and Soft Tissue Morphology Affecting Prognosis and Treatment Planning in Orthodontics*

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The dental profession's preoccupation with the crowns of the teeth and occlusion has retarded our advance in knowledge as to the causes of dento-alveolar abnormalities. This has resulted in empiricism in orthodontic treatment and too much stress on mechanotherapy.

Men like Wingate Todd and Hellman did not sufficiently stimulate the profession's interest in skeletal morphology although their work was followed by that of Brodie and Broadbent. Today we are beginning to apply a still rather limited knowledge of skeletal morphology, its ethnic origin and inheritance, to orthodontics, prosthetics, and periodontal problems. However, for a wider appreciation of the problem the individual must be studied as a functional unit and for this soft tissue morphology and the physiology of posture and behaviour, both innate and learned, must be more fully studied. Thompson has given a lead in one direction with his study on the rest position of the mandible. Rix also stimulated interest in function by his paper on the abnormal swallowing action. In connection with function, Rogers' name should be mentioned. However, in my opinion, his concept was unsound because it assumed that posture and behaviour could be readily changed by function and that function would change form.

*Read at the 13th Australian Dental Congress, Brisbane, June, 1953.

It is the author's belief that a basic concept, not only of orthodontics but of dental science as a whole, is that the dento-alveolar structures, whether normal or abnormal in their position, are in balance in soft tissue behaviour, and have grown from dental bases whose size, position and relationship are in most cases genetically determined, the balance in soft tissue being antero-posteriorly, laterally, and vertically. As Thompson has said, dental science has been hampered by the teaching that the teeth contribute to the height of the face. By teeth he must obviously have meant the dento-alveolar structures.

In spite of the fact that many people still believe that orthodontic treatment will stimulate growth changes — and this particularly applies to those who use functional appliances — no satisfactory evidence has been produced which suggests that the dental base relationships can be changed by treatment, and, in fact, all the evidence is to the contrary. In other words, if at an early age we see an individual with a mandibular dental base posterior to a maxillary dental base from the point of view of dental arch relationship, that skeletal pattern is an inherited morphology and will remain abnormal whatever orthdontic treatment we carry out. Likewise, it has not been demonstrated that normal growth of an individual can lead one to expect any major change in such a dental base relationship. A recent paper by Lande shows that, although gnathion grows forward in relation to the Downs' A point as a normal growth trend, as he points out in his conclusions, the relationship of A to B points hardly changes at all from the age of three to seventeen. Downs' A and B points, I believe, are useful for demonstrating the anterior parts of maxillary and mandibular dental bases.

A cephalometric analysis of our treated cases supports the important conclusions reached by Brodie and others that, with the exception of certain relatively minor postural abnormalities which will be discussed later, all changes that occur as the result of treatment are within the dento-alveolar structure. These are mainly labio-lingual changes of axial inclination of the dento-alveolar structures of the labial segments and antero-posterior movement of buccal segments to maintain the intact arch form. Part of the Tweed concept which to me is a fundamental in treatment planning is an assessment in each case of the stable position of the lower labial segment in the end result. I do not agree, however, that this stable position is "over basal bone"; it is stability in soft tissue behaviour and will be discussed later.

If it is accepted that the dental base relationship cannot change, and that the dento-

alveolar structures are in balance in soft tissue behaviour, then it is important from the point of view of treatment planning and prognosis to know firstly how the soft tissue behaviour has produced abnormalities of dento-alveolar structures or contributes to the abnormality when this is basically the result of abnormal dental base relationship; further, it is important to know how soft tissue behaviour can be modified to stabilise the end result after treatment.

It can be said, then, that the factors producing variations or abnormalities of dental arch relationship can be classified under two headings. They are:

1. The skeletal morphology.

2. The soft tissue morphology and behaviour. Contributing factors are:

(i) Developmental factors within the dento-alveolar structures, the most important of which is tooth size to dental arch size discrepancy.

(ii) Abnormal paths of closure.

In this paper I am mainly concerned with the first two, and, to a lesser extent, abnormal paths of closure.

In the infant and the edentulous individual (Figs. 1 and 2) we see soft tissue and skeletal morphology combining to produce a functional unit, the mandible having a postural relationship to the maxilla as an innate pattern, the intermaxillary space being filled with soft tissue. During development the dento-alveolar structures grow vertically from the dental bases which have a predetermined relationship. The resultant of the forces between tongue and lips and cheeks direct the dento-alveolar structures into a normal or abnormal occlusion. Many discrepancies of dental base relationships, both antero-posteriorly and laterally, are modified to produce acceptable occlusions as the result of this development of the dento-alveolar structures in the soft tissue morphology. Likewise, on many relatively normal dental base relationships abnormal occlusions can be produced by variations of posture and behaviour of the tongue and lips.

As diagnosis and treatment planning is based on labial segment relationship it would be as well to define normal or acceptable incisor relationship. Although this is most easily done from tracings of lateral radiographs I do not wish to infer that the lateral radiograph is a template for assessment of the normal or abnormal. It is, I believe, the best means we have available for demonstrating the skeletal morphology, including the dento-alveolar structure position on that skeletal morphology, to the undergraduate and post-graduate. It is likewise a valuable diagnostic aid. However, as I have already said, a complete examination of the individual as a functional unit is absolutely essential.

Fig. 1.—Lateral radiograph of infant.

In a society which is predominantly Anglo-Saxon, I believe the mean of the normal or acceptable incisor relationship is very much as in figure 3. Firstly, the axial inclination of the upper incisor is given as 107° (+ or − 2.5°) to the Frankfort plane. This figure is a mean value which I calculated myself on 150 normals about eight years ago, and is very nearly that which other investigators have given. What is significant, however, is the angle between upper and lower incisors, mean 135°. From clinical observation there is no doubt whatsoever that any angle above 140° leads to an incisor occlusion which will not maintain a normal overbite (Ballard, 1948). There is an increase of overbite (Fig. 4). Any reduction of the angle below 130° on a normal dental base relationship is a bimaxillary proclination which aesthetically is not accepted by the parents and patient as normal, although more satisfactory than the high angle with excessive overbite.

It is a misconception to think that incisor relationship is dependent on the vertical growth of the cheek teeth. Bjork has pointed out that in positive horizontal overbite or overjet "it is not the degree of prognathism that is the deciding factor, but the relative difference in prognathism between the jaws." Thompson and Brodie have stated that the vertical growth of the dento-alveolar structures does not contribute to face height. It

follows logically from the reasoning that the dento-alveolar structures just fill a predetermined intermaxillary space, that the vertical relationship of the incisors is determined by the normal or abnormal occlusal relationship which is established as a resultant of the dental base relationship and the soft tissue behaviour patterns. If there is no occlusal relationship — in other words, an anterior open bite — then this is invariably the result either of a high Frankfort-mandibular plane angle (high gonial angle) with excessive intermaxillary space anteriorly which complete vertical development of the dento-alveolar structures fails to close, or it is the result of soft tissue behaviour patterns such as a tongue thrust which prevents vertical development of the labial segments into occlusion.

Fig. 2.—Lateral radiograph of edentulous adult.

Many investigators in America have calculated the mean value of the axial inclination of the lower incisor to the mandibular plane. The results vary between 90° + or – 2.5°, and 90° + or – 5°. It is important, however, to remember that if our concept of normal incisor relationships is correct, the axial inclination of the lower incisors to the mandibular plane will vary inversely as the Frankfort-mandibular plane angle (Ballard, Johnson).

If we accept these mean values as giving a concept of the normal it is possible to define what must be the labial segment dental base relationship on the skeletal morphology for the soft tissues to direct vertical development of the labial segment in such a way that a normal incisal occlusal relationship will be produced. Definition could be as follows: with the upper incisors at 107° to the Frankfort plane, and with the lower incisors at 90° to the mandibular plane, and the Frankfort-mandibular plane angle at 27°, then the incisal tip of the lower incisor meets the middle third of the lingual surface of the upper incisor in centric occlusion.

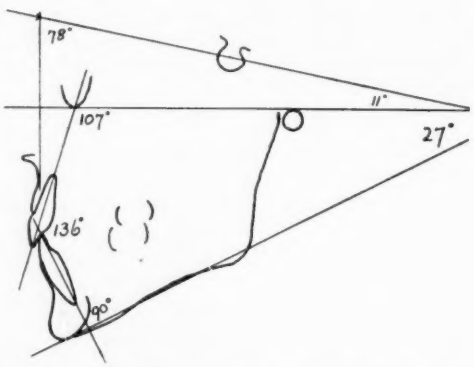

Fig. 3.—Lateral tracing showing mean values.

There is one other factor to be borne in mind when using such a definition for clinical purposes, and that is that the position of the apices of the teeth on the dental base can vary. In spite of this, however, the main reason for adhering to this definition of normal is that from the point of view of post-graduate teaching and clinical work it enables the operator to visualise incisor relationship,

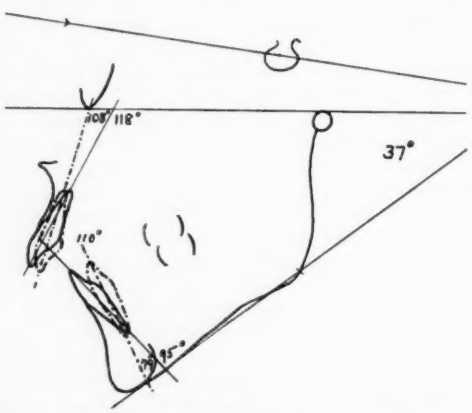

Fig. 4.—Incisor relationships: 1-6 on normal dental base relationship, 7-12 on Skeletal II dental base relationship.

whereas a bare description of dental base relationship from the Downs' A and B points does not.

With this average picture in mind, it is possible with the lateral radiograph of any

case of malocclusion to assess for clinical purposes the antero-posterior relationship of the dental bases. Figure 4 demonstrates the method; it is the tracing of an Angle's Class II, Division 1 occlusal abnormality associated with incompetent musculature and a tongue thrust. For the assessment of dental base relationship the upper incisor outline has been rotated, keeping the apex on the dental base so that it has a correct axial inclination to the Frankfort plane. Similarly, the lower incisor outline has been rotated, again keeping the apex within the dental base, until its axial inclination is correct to the mandibular plane. A Frankfort-mandibular plane angle of 37° is 10° above the mean value, and therefore the lower incisor-mandibular plane angle varying inversely must be 10° below the mean value. The overjet on these re-positioned outlines gives, for clinical purposes, a clear picture of the antero-posterior abnormality of dental base relationship. As it is assumed that this dental base relationship cannot be changed, then this degree of abnormality will affect the prognosis of the case. Figure 5

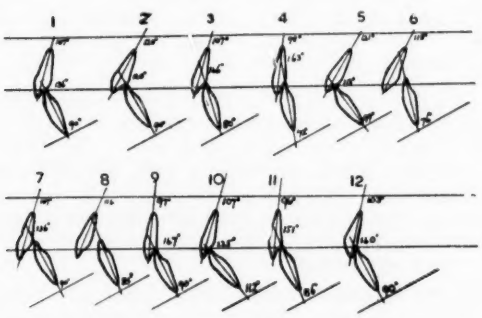

Fig. 5.—Method of assessing dental base relationship with correction of axial inclination of incisors.

(7), (8), (9), (10), (11) demonstrates some of the incisor relationships on a Skeletal II pattern with (10) the most favourable and (9) and (11) unfavourable occlusions after correction of any overjet.

It has been our habit for some years now to classify our dental base relationships in the terminology of Angle's classification; in other words, if the dental base relationship falls within the normal, we call the case a Skeletal I; if the mandibular dental base is posterior to the maxillary base, we call it a Skeletal II; and if the mandibular dental base is in front of the maxillary dental base, it is a Skeletal III.

To illustrate the significance of the two factors under discussion, figure 6(a), (b), (c), shows an extreme Skeletal II dental base relationship with an excessive incisor overjet, the labial segments being in balance in soft tissue behaviour (Fig. 6(a)). The best incisor

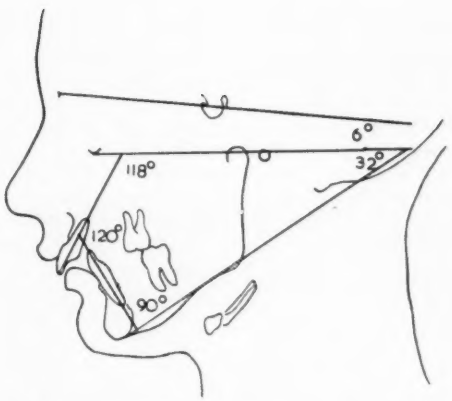

Fig. 6 (a).—Lateral tracing of typical Class II, Division 1.

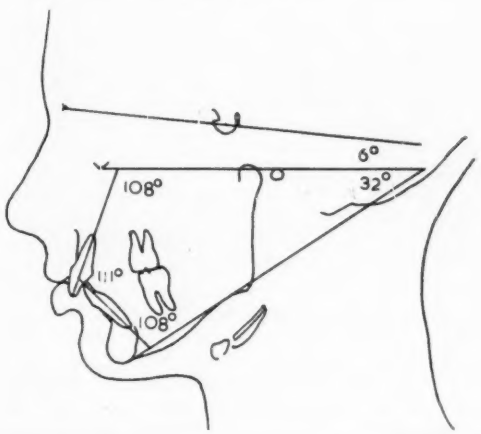

Fig. 6 (b).—Theoretical optimum incisor relationship

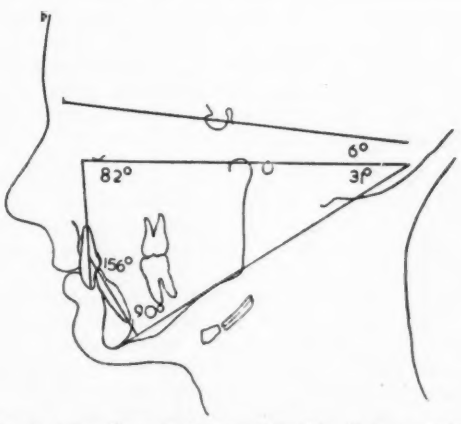

Fig. 6 (c).—Poor incisor relationship if lower labial segment cannot be proclinated.

relationship on this dental base relationship would be achieved by proclinating the lower labial segment many degrees to the upper

incisors, which had been retracted to an aesthetically satisfactory angle (Fig. 6(b)). However, frequently such a proclination of the lower labial segment relapses. This relapse is due to the fact that the lip action will not permit such a proclination. If soft tissue will not permit any proclination of the lower labial segment, then the incisor relationship in the end result will have to be as figure 6(c). This is unsatisfactory, or, as we would say, the prognosis would be poor. On the other hand, as we shall see later, this is unfortunately the only stable end result in many cases, and an attempt to achieve any better relationship is doomed to failure.

Before discussing soft tissue behaviour in greater detail, I must mention briefly the other factors governing dento-alveolar relationships. In the crowded dental arch it must be realised that the arch form is the result of soft tissue behaviour, and that crowding cannot be relieved either by labial movement of incisors or lateral expansion of the arch, without changing the soft tissue behaviour, unless relapse is inevitable. Similarly, a discrepancy between the size of the teeth and the size of the arch can result in a forward position of the buccal segments in relation to the labial segments, and can produce incisor crowding. Distal movement of the buccal segments is usually difficult unless teeth are removed at the back of the mouth. But what is more important in this forward position of the buccal segments is that if the upper labial segment in particular is proclinated in soft tissue behaviour, the general tendency in dental arches, which are a little too small for the teeth, is for the buccal segments to be forward on the dental base and in contact with the labial segment. In such cases it is a question of shifting cheek teeth distally before or during the correction of the proclination of the upper labial segment. This is an important factor to be dealt with from the point of view of anchorage in our treatment planning.

The fourth factor which is concerned in diagnosis is the resting position of the mandible and the path of closure. This has been stated very fully by Thompson, Brodie and Ricketts, and I will only mention the facts briefly. The proprioceptive fibres in the supporting structures of the teeth are very sensitive to abnormal cuspal relationships, and the mandible reflexly swings from such an abnormal contact. It is possible, particularly in a Class II, Division II type of incisor relationship, for the mandible to be taken distally a matter of a millimetre or two to eliminate such an abnormal contact. This distal movement is associated with a contraction of the posterior horizontal fibres of the temporalis muscle. Because there is a muscle contraction at the time when the muscles should be relaxing at the moment of occlusion, there is associated with this distal displacement an excessive freeway space or a true overclosure. Such a distal displacement will, of course, affect dental base relationship to a mild degree when such assessment is made from a lateral skull radiograph taken in occlusion, and must be taken into account both in treatment and prognosis. Similarly, in Skeletal III cases a potential edge-to-edge relationship frequently results in a protrusive movement of the mandible from the rest position. Some orthodontists call this a pseudo-Class III. Again, when there is a lateral abnormality of relationship of the buccal segments, the mandible may swing to left or right to adjust occlusal relationships. For instance, if the maxillary arch is slightly contracted, as it frequently is when there is an abnormal swallow, then the individual forms the habit subconsciously of swinging the mandible to left or right from rest into occlusion to produce a normal lateral relationship of the buccal segments on one side, and a lingual occlusion of the mandibular buccal segment to the maxillary buccal segment on the other.

There is one other postural abnormality of the mandible which must be noted, and which has been previously described (Ballard, Ricketts). When individuals have an extreme overjet they frequently form the habit of posturing the mandible forward the better to overcome the overjet from the point of view of closing the lips.

It will be realised from what has already been said that it is possible to have an abnormal incisor relationship on a normal dental base relationship, the result of abnormal soft tissue behaviour; likewise, if there is an abnormal dental base relationship it is important to know how soft tissue behaviour is related to the abnormality on that abnormal dental base relationship. In both cases it must be known how the soft tissue behaviour can be changed before treatment can be planned to produce a stable end result.

Before discussing abnormalities of soft tissue behaviour it is important to have some picture of the normal. For clinical purposes the normal resting position can be defined as follows: with the mandible in the physiological resting position (Thompson) the lips are in contact with the mimetic muscles at physiological resting tonus, the teeth being apart about 2 mm. — the freeway space. The tongue is arched and in contact with the soft palate, not entirely in contact with the roof of the mouth, with the tip of the tongue either just behind the upper incisors against the roof

of the mouth or sometimes behind the lower incisors when there is a high gonial angle. This posture of the lips is called competent (Ballard, Gwynne-Evans).

An abnormal posture of the lips which in the past has been called the adenoidal facies is that which we now call incompetent lip posture. In these cases this posture is undoubtedly as genetic in origin as the normal just described. The mandible is in the physiological resting posture — there is no abnormal freeway space; the mimetic muscles are at resting tonus, but the lips do not meet, and the individual can only make them meet by a vigorous contraction of the orbicularis oris and mentalis muscles. As has been pointed out in previous papers, it is quite incorrect to call this an adenoidal facies. It is not the result of chronic nasal obstruction, and in the majority of cases these individuals are not mouth-breathers because the tongue and soft palate are in apposition and close off the oral cavity from the oro-pharynx. (Figs. 1, 15(a) and 16.) These individuals from the lips apart posture have to contract the orbicularis oris and mentalis muscles to close the lips every time they swallow, and in some cases they will teach themselves to maintain the lips closed posture. There is also the rather rare condition of posterior oral incompetence, when in resting posture the tongue is not against the soft palate (Fig. 7).

Several years of clinical observation on these individuals have shown quite conclusively that *no* amount of exercising on their part or the application of conscious effort to maintain the lips closed posture ever alters the inherent physiological resting position. They may, after conscious effort, maintain a lips closed posture subconsciously, but it is still by contraction of the orbicularis oris and mentalis muscles. Further, clinical observation has shown that when the incompetence is excessive the muscle effort cannot be maintained; in other words, the individual goes about with the lips apart. Intermediately there are individuals who maintain the lips closed posture for several years and then give up the unequal struggle against inherent patterns and revert to their lips apart resting posture, with the result that if orthodontic treatment was being maintained it will then relapse. The significance of the incompetent lip action to the position of the labial segments is that in overcoming the incompetence it is mainly the orbicularis oris and mentalis muscles in the lower lip which contract. This contraction tends to hold back, as it were, the lower labial segment, the lower lip lifting up underneath the upper labial segment, so proclinating the upper incisors. The result is a Class II, Division 1 incisor relationship. There is not as yet sufficient clinical evidence to say that this will always occur when there is a normal skeletal pattern, but the incompetent lip action will exaggerate a Class II skeletal pattern by increasing the overjet in the way already described. From the point of view of treatment planning the significant

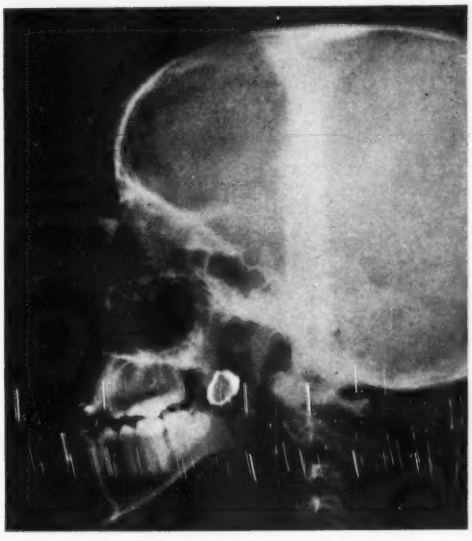

Fig. 7.—Lateral radiograph showing posterior oral incompetence. Soft palate shadow away from tongue shadow.

thing is that if an individual is truly incompetent, and he is to overcome the lips apart posture by contraction of the mentalis and orbicularis oris, then the lower labial segment must *not* be proclinated to help overcome the overjet which is associated with a Skeletal II pattern. The constant contraction of the mentalis and orbicularis oris will cause a relapse. In treatment planning, therefore, anchorage must be so balanced that the lower arch does not move forward, and the lower incisors are not labially inclined.

Case 1. The boy is incompetent, and although there are no pictures taken before the commencement of treatment there has been no change in the soft tissue patterning (Fig. 8). The superimposed tracings (Fig. 9) indicate the tooth movement achieved by empirical Class II traction. This was regarded as a satisfactory result on such a Skeletal II pattern. The boy was fitted with a retainer for a time and given lip exercises. The treatment completely relapsed because of the necessity for contraction of the mentalis and orbicularis oris muscles of the lower lip to overcome the incompetence. He had to be re-treated by the extraction of $\frac{4|4}{}$ and retraction of $\underline{321|123}$. The stable position of the lower labial seg-

Fig. 8 (a).—Case 1: Lips apart resting posture.

ment was, in fact, very nearly the same as that before treatment was commenced (Fig. 10). If this had been appreciated he would have been saved two years' treatment. The present position of the teeth is stable, but not aesthetically entirely satisfactory, the upper incisors being lingually inclined a little too much. He still has a tip of tongue to lower lip resting position, with the same thrust during swallowing against the contracted lower lip, and this helps to maintain an increase of overjet which aesthetically is better than if the upper incisors were taken right back to the stable position of his lower incisors.

Fig. 8 (b).—Case 1: Lips closed, contraction of orbicularis oris and mentalis.

If we adhere rigidly to the definition of competent and incompetent lip action it will

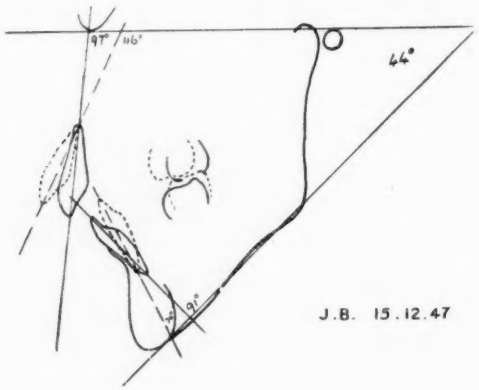

Fig. 9.—Superimposed tracings of Case 1 before and at the end of first period of treatment.

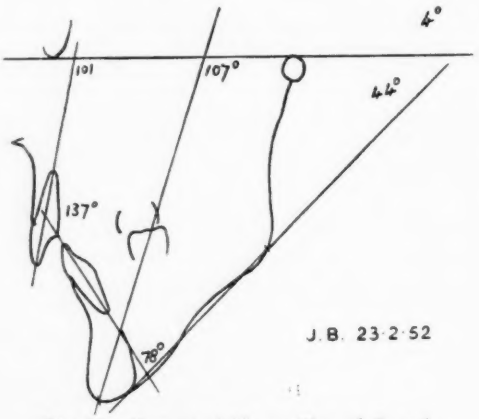

Fig. 10.—Present stable position of Case 1.

be noted that many people without an abnormality have, in fact, slight incompetence of lip action. In other words, there is some contraction of the orbicularis oris and mentalis muscles to maintain a lips closed posture. From the point of view of treatment planning it is important but difficult to assess whether the lips are incompetent when there is an extreme Skeletal II pattern; the lower lip being related to the mandible it tends to be behind the vertical development of the upper

labial segment, unless the tongue so balances the lower lip that there is a mandibular labial segment proclination. In an extreme Skeletal II, therefore, the lower lip tends to be behind the upper incisors without any contraction of mentalis and orbicularis oris muscles. In such cases the prognosis and treatment planning is entirely different.

Case 2 (Fig. 11). This boy has a Skeletal II dental base relationship but with competent lip action, his lower lip being behind the upper incisors because of the skeletal morphology and not because of lip incompetence. On such a Skeletal II pattern, as has already been said, the most satisfactory

Fig. 11.—Case 2: Lips apart with upper incisors resting on lower lip (full face).

aesthetic and functional incisor relationship is achieved by retracting the upper incisors until they are at an aesthetically satisfactory angle, and then proclinating the lower labial segment to them, bringing the lower buccal segments forward to support this proclination. Because this boy's lip action was competent, proclination of the lower labial segment was possible. Fig. 12 (a) and (b) show before and after tracings, and Fig. 13 before and after models. The lower labial segment has been proclinated from 92° to 102°. This position is stable.

Second only to abnormal dental base relationship in the production of abnormal dento-alveolar relationship are the abnormal behaviours of lip and tongue during swallowing and to a lesser extent during articulation. The abnormal swallow and its definite connection with malocclusion was first described by Rix. A brief description of the mechanism of the normal swallowing action in the oral cavity is as follows: the mandible is held firmly in a position which is just closed from the physiological rest position. With a normal freeway space this could also be described as in light occlusion, but with an excessive freeway space the cheek teeth would be apart. From the mandible so fixed the mylo-hyoids contract and lift the tongue; simultaneously

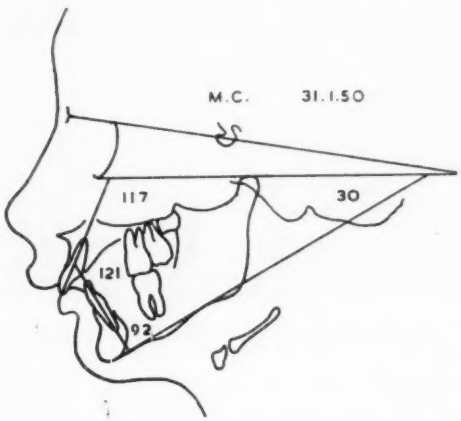

Fig. 12 (a).—Lateral tracing before treatment of Case 2.

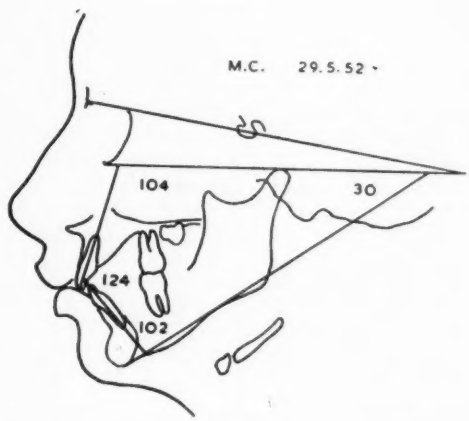

Fig. 12 (b).—Lateral tracing after treatment of Case 2.

the tip comes up against the palate behind the upper incisors as the commencement of the peristaltic wave which passes the food backwards into the oro-pharynx. Because the tongue performs these movements in the rigid walled oral cavity the lips and cheeks play no part.

In the abnormal swallow, on the other hand, the anterior border of the tongue does not commence the peristaltic wave by flipping up against the palate, but is pushed forward as a rule against the lips, the lower in particular.

The orbicularis oris and mentalis muscles contract to resist this thrust. In many individuals with the abnormal swallow there is also a lower lip against tip of tongue resting posture. In such cases there is reflex contraction of orbicularis oris and mentalis to balance the tongue thrust.

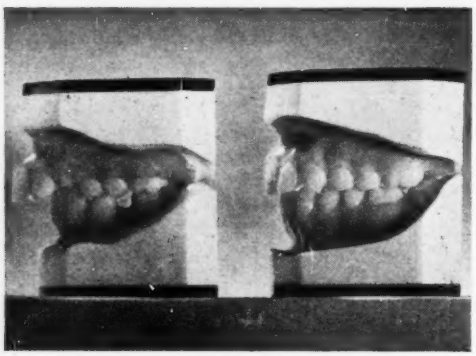

Fig. 13.—Before and after models of Case 2.

The deformity of the dento-alveolar structures which results from this abnormal swallow appears to vary according to (1) the dental base relationship, (2) the basic position, as it were, of the labial segments in soft tissue behaviour on which has been superimposed the abnormal swallow. To elaborate, there must be space between upper and lower incisors through which the tongue can thrust. If there would have been an overjet as a result of other factors then the tongue thrusts against the upper incisors, proclinating them; the lower lip contracts against the tongue thrust, so holding back the lower labial segment, the total effect being to increase the overjet.

On the other hand, if the abnormal swallow is superimposed on a skeletal pattern and soft tissue morphology which would produce a normal overjet, then the tendency is for the tongue thrust to produce a reduced overbite or open bite.

Cases 3 *and* 4 (Figs. 14 and 15). The patients are sisters, both of whom had abnormal swallowing action with tip of tongue to lower lip resting posture and an interdental sigmatism. It will be seen from the lateral skulls, however, that similar behaviour patterns have been superimposed on different skeletal morphologies and different basic positions of the lower labial segments. In figure 14 the position of the lower labial segment is proclinated, which compensates for the Skeletal II pattern to a certain extent, reduces the overjet, and so there is a marked open bite as a result of the tongue thrust. On the other hand, in figure 15 the position

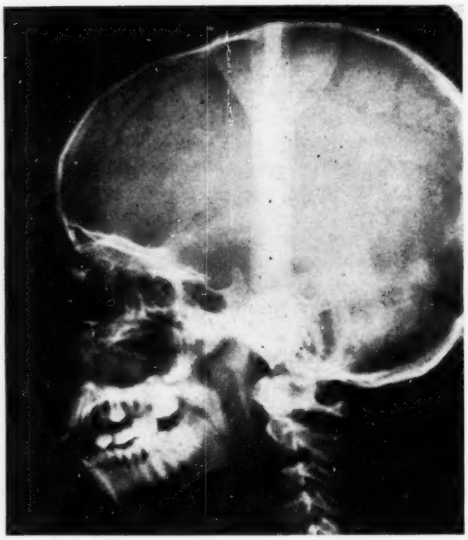

Fig. 14 (a).—Case 3: Lateral radiograph shows elevated soft palate.

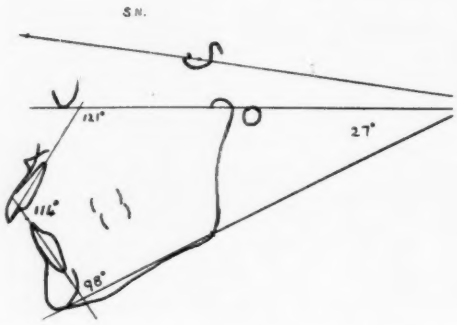

Fig. 14 (b).—Case 3: Lateral tracing shows her relatively proclinated lower labial segment.

of the lower labial segment is relatively lingually inclined with the tongue thrust producing more of an increase of overjet than of open bite. Prognosis in Case 3 is good if the tongue thrust can be eliminated and treatment must be planned to achieve distal movement of the maxillary teeth to the present position of the lowers. Prognosis in Case 4 depends on some proclination of the lower labial segment being stable as well as elimination of the tongue thrust. Treatment must be planned, therefore, to take back the maxillary arch and bring forward the mandibular arch. It is interesting to note that the lateral radiograph of Case 3 shows an elevated soft palate which was the result of her swallowing as the x-ray was taken.

As has been said, on many cases of abnormal swallowing there is also an abnormal resting behaviour, in which the tip of the tongue is forward in contact with the lower

lip, the orbicularis oris and mentalis muscles being slightly contracted. This posture is also a factor in preventing full vertical development of the lower labial segment, and therefore produces a reduced overbite.

There is no time for a lengthy and philosophical discussion on the aetiology of the abnormal swallow, but a plain statement on the trend of our thoughts can be made.

1. It is frequently a characteristic behaviour of the maternal or paternal side of the family.

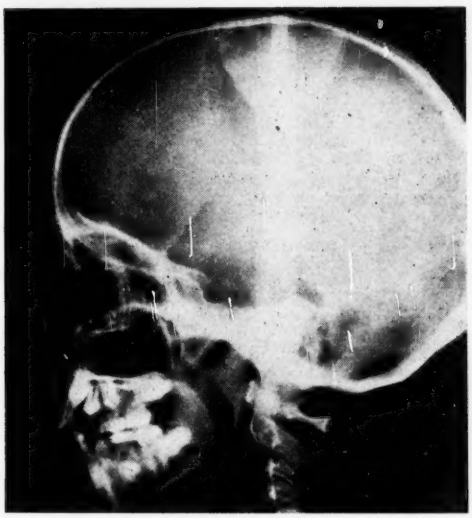

Fig. 15 (a).—Case 4: Lateral radiograph shows shadow of tongue and soft palate in contact.

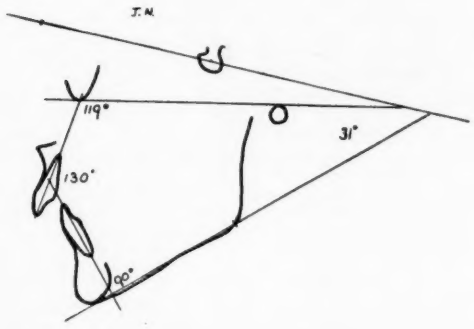

Fig. 15 (b).—Case 4: Lateral tracing shows no proclination of the lower labial segment.

2. Swallowing has to be present at birth as a mature pattern of behaviour, and our observations to date indicate that there is little difference between the tongue movements in infant feeding and adult swallowing (Gwynne-Evans).

3. If the first two statements are correct, then the abnormal swallow is an innate behaviour and not a habit.

4. To support this is the finding that in most cases it is very difficult to eliminate completely an abnormal swallow.

5. The cases with the worst prognosis are those in whom there is an associated interdental sigmatism. In these cases the speech therapist may or may not eliminate the sigmatism, but the tongue thrust is rarely improved at all.

Case 5. Figure 16 is the lateral radiograph of a young man who had several years' orthodontic treatment directed towards closing up his anterior open bite. Associated with the open bite there was abnormal swallowing action, lip to tip of tongue resting posture, and an interdental sigmatism. Even with several years' conscious effort on the part of the patient, the interdental sigmatism and the abnormal swallowing action could not be controlled, and therefore the anterior open bite could not be closed up. The net result of the persistent attempts at treatment was that there was marked absorption of the apices of upper incisors.

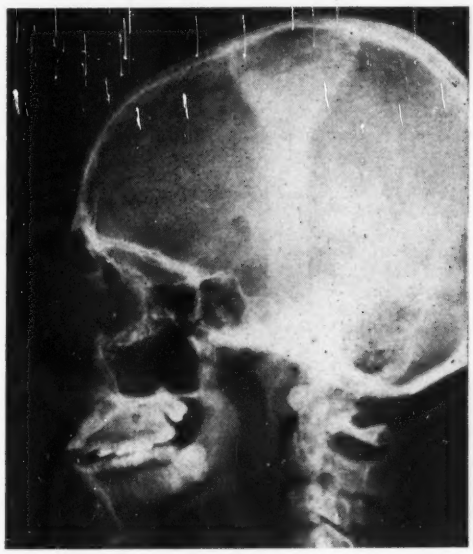

Fig. 16.—Lateral radiograph of Case 5 showing the anterior open bite and absorbed roots of the upper incisors.

This has been a very brief survey of the factors of skeletal and soft tissue morphology as it affects prognosis. It is quite evident that only by careful clinical observation can a more definite knowledge of the relationship between the various postures and behaviours of the facial musculature be obtained. The various types that have been outlined can, of

course, occur in relation to one another and in varying degrees, and therefore a clinical assessment of each individual case is essential in diagnosis and treatment planning. If the stable end result can be assessed before tooth movement is commenced, relapses will be eliminated and prolonged retention will be unnecessary. In fact, the use of prolonged retention is an indication that the orthodontist is not certain of the stability of his case.

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Editorial

Paradontal Disease

"The most common cause of loss of the teeth is the negligence of those people who do not clean their teeth when they might and that they perceive the lodgement of this foreign substance (tartar) which produces disease of the gums."

— Fauchard, 1746.

It is not often that periodontal disease receives adequate recognition, either within the profession or without. This is not because the disease has only recently been recognised or has a limited incidence. The ancient Egyptians observed the clinical condition of teeth with advanced periodontal pathoses and advised cures for them, and the Father of Modern Dentistry, Pierre Fauchard, gave very careful descriptions of gingival conditions. Nor has it a limited incidence because it is claimed that more teeth are lost due to periodontal disease than dental caries.

The apparent lack of interest in periodontal disease is a sad commentary on our approach to dentistry—that of a reparative and restorative health service rather than an essentially preventive outlook. There is little doubt that periodontal diseases are responses to injury of the supporting structures of the teeth and, although the anatomic arrangement of these tissues cause certain specific local changes, the basic pathologic conditions are similar to those in other parts of the body — inflammation, degeneration, atrophy, hypertrophy and hyperplasia. Thus it follows that the understanding of the pathology of the condition is essential to the prevention and treatment of periodontal disease.

However, the problem is further complicated by the effects of systemic factors, for, as Bunting has suggested,

> . . . disturbances in the general health and nutrition of the body frequently produce marked changes in the tone and vital resistance of the gingival and periodontal tissues and render them more susceptible to injury from traumatic stresses and bacterial invasion.[1]

Although there has been considerable research, both clinical and histo-pathological, periodontal disease is still a controversial subject. In spite of the large body of scientific information obtained by reliable investigators, such aspects as the mechanism of the epithelial attachment and of pocket formation and deepening, the relation of systemic disease to periodontitis and continuous passive eruption of the teeth are still problems without adequate solution.

In spite of the lack of agreement on many phases of periodontal diseases, clinical experience indicates that 80% or more of cases are caused by local factors which injure the gingival and periodontal tissues, a whole range of irritants, calculus, faulty dentistry, food impaction, chemical irritants and dysfunction.

In this issue of the Journal there is presented a comprehensive practical approach to periodontal treatment.

Progress in diagnosis and treatment planning depends on the facts covered by clinical research and laboratory investigations. However, for the present, clinical experience enables a wide range of successful periodontal therapy to be utilized.

It is our responsibility to make the benefits of periodontal treatment available to our patients, for with the increasing control of dental caries and the problem of an ageing population the future patient will reach middle life with a fuller complement of natural teeth, caries-free, and accordingly the supporting structures of the teeth will be the focus of dental attention.

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News and Notes

Federation Dentaire Internationale

REPORT ON THE XLIST ANNUAL MEETING AT OSLO, NORWAY, 26TH JULY-1ST AUGUST, 1953.

The XLIst Annual Meeting of the Federation Dentaire Internationale was held in Oslo, Norway, from 26th July to 1st August under the auspices of the Norwegian Dental Association.

This was the first Annual Meeting of the F.D.I. since the adoption of the new Constitution in London last year, when radical changes were made in the structure and framework, and this meeting marked the beginning of this new era in the affairs of the Federation.

The meeting was therefore of particular interest to the Member Associations who were anxious to see how the new machinery would function under the new Constitution. It must be said at once that the meeting was an undoubted success, thanks to the vision and hard work of the Norwegian Organising Committee under the leadership of Dr. Grythe, the Chairman, ably assisted by his Committee, with Dr. Gudvin Sydnes as Secretary and Dr. Knut Gard as Treasurer. The indefatigable work of these men made it possible to proclaim the meeting as one of the most successful in the annals of the Federation.

H. R. Sullivan Appointed Editor of Journal

Dr. Harold Sullivan has recently been appointed to the editorship of *The Dental Journal of Australia* for a two year period, 1954-55.

Dr. Sullivan is at present Assistant Director of the Institute of Dental Research and a part-time Lecturer in the Faculty of Dentistry. The Senate of the University of Sydney this year conferred on him a Doctorate of Dental Science for his research in the problem of the initiation of dental caries.

Dr. Sullivan was awarded a Nuffield Scholarship for 1952 and spent that year in England engaged in research work. Before that time he had taken an active part in Association affairs and served as a member of the Executive of this Branch of the Association. Dr. Sullivan was also a member of the committee which prepared the first report on fluoridation for the Association in 1947.

In 1951, Dr. Sullivan was Honorary Editor of the Journal before his departure overseas.

The Executive and members have every reason to be confident of the future of the Journal in the appointment of Harold Sullivan as Editor, for his exceptional experience and academic record fit him admirably for the task.

Albert Joachim International Prize

On the recommendation of the Association Generale des Dentistes de Belgique, the Belgian Dental Profession wished to commemorate the 50th professional anniversary of Dr. Albert Joachim, one of the most eminent members of the dental profession in Belgium.

In accordance with the express wish of Dr. Joachim, a Foundation to encourage scientific research in Odonto-Stomatology has been formed and the Administrating Committee of this Foundation was of the opinion that one of the best ways of fulfilling its aims was to award a prize for research work.

It was decided to allocate a large proportion of the funds collected to a national and an international prize.

The Federation Dentaire Internationale at its Annual Meeting in Oslo, July, 1953, decided to accept the invitation of the Foundation Committee to be responsible for the awarding of the International Prize, in consideration of the eminent service rendered on an international plane by Dr. Albert Joachim, who for forty years has been the Belgian representative to the Federation showing at all times unremitting interest and co-operation.

Dr. Joachim, who is an F.D.S.R.C.S. (Eng.), is one of the Vice-Presidents of the Federation Dentaire Internationale and it gives the Federation great pleasure to join in the tribute given to its eminent colleague.

The first award of the Albert Joachim Internationale Prize, which will be made at the XIIth Internationale Dental Congress in Rome, 1957, will amount to 50,000 Belgian francs.

It will be awarded by a Jury appointed by the Council of the Federation Dentaire Internationale for the best purely scientific research in the field of Odonto-Stomatology.

Attention is particularly drawn to the fact that only strictly *original* work will be considered, that is personal research by the author which has not yet been published.

Since the General Assembly of the Federation Dentaire Internationale has asked the Council of the Federation to make certain amendments to the rules of the competition, these will have to be published later.

Anybody interested in the prize can, for the moment, write to the Secretary General of the Federation, Dr. G. H. Leatherman, 35 Devonshire Place, London, W.1, England, who will give them all the information they require.

Professor Arnott Appointed Fellow of Senate for Further Period

It is gratifying to note that Professor A. J. Arnott, Dean of the Faculty of Dentistry, University of Sydney, has been appointed a Fellow of the Senate of that University to hold office for a further period of two years from 1st January, 1954. It will be remembered that Professor Arnott was appointed a Fellow of the Senate in January, 1952, and occupied this office for the past two years.

Obituary

William Harcourt Andrews.

William Harcourt Andrews, a very respected practitioner who recently retired from practice in the city of Sydney, passed away suddenly in Auckland on 17th December, 1953.

Mr. Andrews was one of the original members of the New South Wales Branch. He served in the first War in charge of the laboratory service at the Dental Hospital, finally achieving the rank of Captain, A.A.M.F.

Deepest sympathy is extended to his widow.

Jennings Shield

The 18th Annual Cricket Match between the Dental Profession and the Dental Undergraduates for the Jennings Shield took place on 10th December in ideal weather on the picturesque University Oval. On a time limit of 2¼ hours each, the undergraduates won by scoring 196 for 6 wickets against the dentists' score of 170 for 7 wickets, both sides playing entertaining cricket.

Trophies were presented to the best bat, bowler and fielder of each team, i.e., Messrs. K. Binns, R. Stewart and A. Vern Barnett for the Dentists and Messrs. W. J. Mackie, F. Jones and J. Fahey for the Undergrads.

Appreciation was expressed by Mr. R. Norton, to which Mr. John T. Jennings responded.

Australian Red Cross Society March Appeal, 1954

"PLAYING THEIR WAY TO HEALTH"

There is an old saying, "I used to grumble because I had no shoes until I met a man who had no feet," and it is often true that people with real handicaps are more cheerful and uncomplaining than those with minor illnesses. Certainly it is true at the Red Cross Lady Lawley-Cottage-by-the-Sea in Western Australia.

Tiny children with twisted limbs often spend years at the Cottage undergoing treatment, and yet there is little misery among the patients. The keynote of the home is happiness and co-operation: this partly because re-education of the limbs is a "game" and everyone is so kind and helpful, and partly because all the children suffer from some handicap and learn to help each other in the fight for a normal life.

There are 38 beds at the Cottage, which has been maintained for some years by the West Australian Division of the Society at considerable expense. This year, the Cottage has been classified as a Class C Hospital, which will mean some financial help through the Commonwealth Government Health Scheme.

Patients include medical and surgical convalescents and spastics. Most of them are long-term cases, some having been at the Cottage as long as six years. Many are from country areas where part-time physiotherapy treatment is not available.

Staff consists of Matron Young, two sisters, a physiotherapist, and assistant nursing and domestic staff. A Speech Therapist visits the Cottage and the Education Department provides two school teachers.

The Society provides clothing, board and lodging, nursing attention, physio- and speech therapy and surgical aids. Handicraft workers conduct classes among the patients.

This is one of many services conducted by YOUR Red Cross. To continue this work Red Cross needs YOUR HELP!!!

Jocelyn has left her crutches aside while she concentrates on her school work. Behind her are gaily painted wall pictures to brighten the class room.

Association Activities

Australian Dental Association (New South Wales Branch)

General Meetings.

Additional General Meeting.

An additional Ordinary General Meeting of the Association was held on Tuesday, 17th November, 1953, in order to enable members to hear an address by Professor Basil Bibby, Ph. D., D.M.D., of New York, U.S.A.

Professor Bibby, who was visiting Australia at the invitation of the Australian Dental Association under the auspices of the United States Educational Foundation in Australia, was a most welcome visitor and lecturer. His address, entitled "Dental Research and Caries Control—Its Influence on Private Practice," was most appreciated by the large audience of members present.

Annual General Meeting.

The Annual General Meeting of the Association was held on Tuesday, 24th November, 1953.

Annual Report, Treasurer's Report and Appointment of Auditor.

The Annual Report of the Executive, the Treasurer's Report, Accounts and Balance Sheets, and the Auditors' Report for the financial year ended 31st August, 1953, were presented to the meeting and adopted.

The meeting appointed Messrs. C. O. Beck & Wayland Auditors of the Association for the ensuing year.

Honorary Members for 1954.

The meeting elected the following 15 persons as Honorary Members for the year 1954:

President, Australian Dental Association; President, American Dental Association; President, British Dental Association; President, New Zealand Dental Association; President, British Medical Association (New South Wales Branch); President, Dental Board of New South Wales; Professor A. J. Arnott; Dr. P. C. Charlton; Dr. N. E. Goldsworthy; Mr. T. T. Alkin; Sir Harry Moxham; Mr. Keith Oatley; Mr. G. T. Chauvel; Professor E. Ford; Mrs. John Barr.

Election of Executive.

The ballot for the election of the Executive of the Association for the ensuing year resulted in the following persons being announced to the meeting as elected:

Mr. G. E. Baulman; Mr. N. E. Edney; Mr. H. McD. Finnie; Mr. J. G. Fletcher; Mr. E. J. Gee; Mr. W. A. Grainger; Dr. P. B. Green; Dr. F. E. Helmore; Mr. A. G. Hunter; Mr. R. Krauss; Dr. A. G. H. Lawes; Mr. R. G. Leeder; Mr. R. Y. Norton; Dr. A. G. Rowell; Mr. T. Royse-Smith; Mr. R. Tompson.

Ordinary General Meeting.

Following the closure of the Annual General Meeting on 24th November, 1953, an Ordinary General Meeting of the Association was convened. Three films recently obtained from the United States Navy were screened and viewed by members.

During the meeting the newly elected Executive, who had retired for the purpose of electing the Officers of the Association for the ensuing year, returned and were formally presented to the meeting. The Officers of the Association for the ensuing year were then announced by the Chairman, such Officers being:

President: Dr. F. E. Helmore; Vice-Presidents: Mr. W. A. Grainger and Dr. A. G. Rowell; Hon. Treasurer: Mr. J. G. Fletcher.

The newly elected President of the Association, in addressing the meeting from the Chair, conveyed the appreciation of the Executive and members of the services of the retired Officers and Executive. He paid special tribute to the untiring services to the Association of the Immediate Past President, Mr. N. E. Edney, who suitably responded.

Executive Report.

Election of Officers.

As reported above, the first duty of the newly elected Executive at its initial meeting was to elect the Officers of the Association.

Appointment of Additional Members of the Executive.

The newly elected Executive has appointed the following additional members upon the nomination by the respective Divisions under the terms of Article 25A:

Blue Mountains Division: Mr. J. H. Anker; Far North Division: Mr. H. W. Shand; Newcastle and Hunter River District Division: Mr. T. Emanuel; North East Division: Mr. F. C. Haddan; North and North West Division: Dr. B. Martin; South Coast Division: Dr. J. D. Oddy; Southern Division: Mr. A. Broughton; Southern Tablelands Division: Mr. L. Marshall; Western Division: Mr. L. B. Hume.

Appointment of Committees:

The Executive has appointed the following Committees:

Honorary Officers' Committee: Dr. F. E. Helmore (Chairman); Dr. A. G. Rowell, Mr. W. A. Grainger, Mr. J. G. Fletcher, Mr. N. E. Edney.

Defence Committee: Dr. E. R. Magnus (Chairman); Dr. F. E. Helmore, Mr. N. E. Edney, Mr. C. C. Croker, Mr. J. G. Fletcher.

Journal Committee: Mr. A. G. Hunter (Chairman); Mr. G. Baulman; Mr. E. J. Gee, Mr. N. D. Martin, Dr. L. E. McDermott, Mr. R. Y. Norton; Mr. R. Tompson.

Dental Health Committee: Mr. T. Royse-Smith (Chairman); Dr. O. Basil-Jones, Dr. P. B. Green, Mr. N. D. Martin, Mr. B. W. Maundrell, Dr. R. McMullin, Mr. J. Noble, Mr. J. W. Skinner, Mr. R. Tompson, Mr. G. Morse Withycombe.

Research Committee: Mr. W. A. Grainger (Chairman); Professor A. J. Arnott, Dr. D. Freeman, Dr. R. M. Kirkpatrick, Mr. N. D. Martin.

Ethics Committee: Mr. H. McD. Finnie (Chairman); Mr. G. E. Baulman, Mr. J. G. Fletcher, Mr. E. J. Gee, Mr. R. Krauss, Mr. R. G. Leeder.

Syllabus Committee: Mr. E. J. Gee (Chairman); Dr. D. Freeman, Dr. P. B. Green, Dr. A. G. H. Lawes, Dr. L. E. McDermott.

Divisions Committee: Mr. R. Krauss (Chairman); Mr. J. H. Anker, Mr. A. Broughton, Mr. L. B. Hume, Mr. L. Marshall, Dr. J. D. Oddy.

Sports and Social Committee: Mr. R. Y. Norton (Chairman); Mr. K. O. Binns, Mr. E. P. Cordaiy, Mr. F. Dennett, Mr. J. B. Fox, Mr. J. G. Fletcher, Mr. L. J. Noone, Mr. F. R. Reid.

Benevolent and Provident Fund Committee: Mr. J. G. Fletcher (Chairman); Mr. R. C. Dennett, Dr. M. Halliday, Mr. R. Krauss, Mr. L. J. Noone, Dr. J. D. Oddy, Mr. F. R. Reid.

Library Committee: Mr. A. G. Hunter (Chairman); Dr. A. Bull, Dr. C. H. Graham, Dr. R. W. Halliday, Mr. N. D. Martin, Mr. Ralph Tompson, Mr. A. O. Watson.

Post-Graduate Courses Committee: Mr. W. A. Grainger (Chairman); Dr. G. V. Gengos, Dr. R. W. Halliday, Dr. F. E. Helmore, Mr. R. Krauss, Mr. N. D. Martin, Dr. R. N. McMullin.

Committee on Articles of Association: Dr. A. G. Rowell (Chairman); Mr. J. G. Fletcher, Mr. E. J. Gee, Mr. R. Krauss, Mr. R. Y. Norton.

Suburban Dental Organisations Liaison Committee: Dr. F. E. Helmore (Chairman); Mr. N. E. Edney, Dr. A. G. H. Lawes, Mr. E. P. Cordaiy, Mr. R. W. Wilson, Mr. R. Dennett, Mr. R. Hawthorne, Mr. T. Ginty, Mr. C. Sharpe, Mr. L. Noone, Mr. J. Spencer.

Consultative Sub-Committee on National Dental Health: Dr. F. E. Helmore (Chairman); Mr. J. G. Fletcher, Mr. W. A. Grainger.

Appointment of Editor of "The Dental Journal of Australia."

The Executive has been pleased to appoint Dr. H. R. Sullivan as Editor of *The Dental Journal of Australia* for two years as from 1st January, 1954.

The Executive also wishes to record its appreciation of the services rendered by Mr. N. D. Martin as Honorary Editor of *The Dental Journal of Australia* over the last two years. In expressing its appreciation, the Executive congratulates Mr. Martin on the maintenance of the high standard of publication achieved frequently in the face of considerable difficulties.

Hardwick Memorial Library.

The following books have recently been purchased for the Hardwick Memorial Library:

Levy, A Textbook for Dental Assistants.

Montgomery, Pathology for Students of Dentistry.

MEMBERSHIP.

Full Members.

Cairns, Kevin Michael, B.D.S.; Conquest, John, B.D.S.; Grogan, William John, B. D. Sc.; Morris, Alwyn Laurence, B.D.S.; McCarthy, Kevin, B.D.Sc.; Pierson, Terence Edward, B.D.S.

Full Members (previously Qualified).

Currie, Ian A. G.; Macansh, J. D.

Resumption of Full Membership.

Wood, Philip B., B.D.S.

Qualified Members (previously Full).

Barnard, Hal J., B.D.S.; King, Wallace, B.D.S.

Restricted Members (previously Full).

Moir, S. J.; Sainsbury, Arthur William.

Resignations.

Bertram, C. C.; Craig, R. M.; Molony, I. G.; Roseby, B. D.; Rowell, A. E.; Turner, B. M.; Underwood, H. T.; Wallman, J.

Divisional Activities

BLUE MOUNTAINS DIVISION.

The following were elected to the Committee of the Blue Mountains Division for the year 1954:

Chairman: Mr. D. W. McEwan.

Vice-Chairman: Mr. D. Droulers.

Hon. Secretary: Mr. J. Weingarth.

Hon. Treasurer: Mr. W. Hill.

Committee: Mr. P. Clark, Dr. K. Hutchinson, Mr. M. Lumley.

Nominee as Additional Member of the Executive: Mr. J. H. Anker.

FAR NORTH DIVISION.

At the Annual General Meeting of this Division held in Lismore on 21st November, 1953, the following office-bearers were elected for 1954:

Chairman: Mr. H. W. Shand.
Vice-Chairman: Mr. W. H. Scotton.
Hon. Secretary: Mr. J. A. Bice.
Hon. Treasurer: Mr. K. M. Dagg.
Committee: Mr. L. F. Hayley, Mr. T. T. Pearson.
Nominee as Additional Member of the Executive: Mr. H. W. Shand.

NEWCASTLE AND HUNTER RIVER DISTRICT DIVISION.

At the Annual General Meeting of this Division held on 19th November, 1953, the following office-bearers for 1954 were elected:

Chairman: Mr. R. Krauss.
Vice-Chairman: Mr. R. H. Bruce.
Hon. Secretary: Mr. B. C. Dyson.
Hon. Treasurer: Mr. D. L. Reynolds.
Committee: Mr. H. A. Skinner, Mr. G. R. Solomon, Mr. J. W. Todhunter.
Nominee as Additional Member of the Executive: Mr. T. Emanuel.

NORTH AND NORTH WEST DIVISION.

At the Annual General Meeting of the North and North West Division held on 21st November, 1953, the following officers of the Division were elected:

Chairman: Mr. F. L. James.
Hon. Secretary: Mr. T. H. Boshier.
Hon. Treasurer: Mr. A. A. Campbell.
Committee: Dr. J. S. Northcott, Mr. W. M. Lyndon, Mr. C. Heard, Mr. M. Hook.
Nominee as Additional Member of the Executive: Dr. Brian Martin.

SOUTH COAST DIVISION.

The Annual Meeting of this Division was held on Tuesday, 17th November, 1953. The following are the Officers elected for 1954:

Chairman: Mr. C. W. Stone.
Vice-Chairman: Dr. H. C. Maldon.
Hon. Secretary: Mr. J. H. Palmer.
Asst. Secretary: Mr. Derek Maguire.
Hon. Treasurer: Mr. G. Meldrum.
Nominee as Additional Member of the Executive: Dr. J. D. Oddy.

SOUTHERN DIVISION.

At the Annual Meeting of the Southern Division held on Saturday, 24th October, 1953, the following officers were elected:

Chairman: Mr. Archer Broughton.
Vice-Chairman: Mr. J. B. McTaggart.
Hon. Secretary: Mr. J. G. Blakeney.
Hon. Treasurer: Mr. L. O. Caldwell.
Committee: Mr. R. Phillips.
Nominee as Additional Member of the Executive: Mr. A. Broughton.

WESTERN DIVISION.

At the Annual General Meeting of the Division held on 14th November, 1953, at Orange, the following office-bearers and committee were elected:

Chairman: Dr. Dent.
Vice-Chairman: Mr. L. B. Hume.
Hon. Secretary: Dr. J. W. Meldrum.
Hon. Treasurer: Mr. A. C. Dalziel.
Committee: Mr. H. T. Burrows, Mr. K. Campbell, Mr. R. S. Edwards.
Nominee as Additional Member of the Executive: Mr. L. B. Hume.

At this meeting the forthcoming Convention in September, 1954, was discussed and the following members were elected to the Convention:

President: Dr. Dent.
Vice-President: Mr. H. T. Burrows.
Hon. Secretary: Dr. J. W. Meldrum.
Hon. Treasurer: Mr. A. C. Dalziel.
Director of Clinics: Mr. Rupert Edwards.

New Books and Publications

EXPERIMENT IN DENTAL CARE, by J. T. Fulton, Geneva, 1951, World Health Organisation. (87 pp.). *Our copy by courtesy of the publishers.*

The New Zealand School Dental Nurse Service has been the subject of much investigation by Government organisations, professional associations and individuals.

Some countries, notably Indonesia, Malaya and Ceylon, have seen what is done, and, as a result, have taken steps to set up similar institutions.

The United Kingdom Mission made an intensive but short study of the New Zealand Service. The American Dental Association sponsored its own investigation by Gruebbel, which was carried out at a time not far removed from Fulton's study.

The service has not unnaturally been the subject of much controversy amongst dentists the world over because it delegates completely and trustingly the bulk of dental care of young children to women trained in two years to carry out certain technological services related to operative dentistry.

Prior to Fulton's report, much argument based on supposition, opinions and impressions, had been bandied about and what Fulton has done, and done well, is to determine certain facts which would enable an intelligent evaluation of the project.

The study covered a forty day period and was prepared at a meeting of various consultants in Washington. It is to be expected, therefore, that a careful examination has been designed, and it should be noted that this study deals with the caries control measures being used by the Service and their effectiveness as demonstrated in the mouths of a representative group of school children.

The reader will not be disappointed.

It does not measure the state of health of the supporting tissues of the mouth or the extent of malocclusion. No attempt was made to evaluate the programme in terms of what is ideal care for the individual child, nor the results of the programme beyond the age of 14 years.

An important aspect of any survey is the sampling of the subject matter and geographical distribution with population intensity clearly becomes the yardstick in any brief survey. Such matters having been determined, the examinations were commenced and in the forty day period 4,000 children's mouths surveyed.

Teeth were designated according to the conventional D.M.F. index and further classed X, number indicated for extraction, O, number actually missing.

As far as the deciduous teeth were surveyed, the findings were listed de, for the number of molars which were untreated or both decayed and filled, f, filled teeth.

From the examinations, various statistical presentations are given and are dealt with in Chapters 2-4.

Chapters are devoted to a dissection of the costs of the service, the course of training for dental nurses, the dental profession and the development of the service.

A series of ten annexures provide information in the report on such phases of the service as official pronouncements to the staff, Dental Benefits Forms and samples of examinations set down for the trainee dental nurses taking primary, intermediate, and final examinations.

For a limited number (38) of cases, Fulton obtained bite-wing radiograms and used these to determine the standard of proximal contours. The quality of the reproductions suffers in the presentation and numbers are mounted carelessly.

The report contains twelve figures and ten tables and has adequate bibliographical support.

Within the aim, the author has completely covered the requirements of the study and has given the results in an intelligent and unbiassed manner, the controversy and argument has been left to those whose desires lie in that direction.

It is an excellent study and serves clearly to demonstrate the valuable work of the World Health Organisation, a specialised agency of the United Nations.

—Robert Harris.

1952 YEAR BOOK OF DENTISTRY, ed. by S. D. Tylman and others, Chicago, 1953. Year Book Publishers. (519 pp., 316 illus.). *Our copy by courtesy of the publishers.*

This excellent publication maintains the usual high standard established by preceding Year Books.

The fields covered are diagnosis, pulpal and periodontal diseases, caries, public health dentistry for children, orthodontics, surgery and related pathology, restorative and prosthetic dentistry. The section on diagnosis includes radiography and treats advances such as panographic radiography and the long cone technique.

The comments by the editors are invaluable and aid in assessing the value of the contributions.

This book which so successfully summarises current dental thought should be studied by every dentist.

—N. D. Martin.

THE NEWER KNOWLEDGE OF HYGIENE IN DIET, by J. Sim Wallace, Brooklyn, N.Y., 1952. Dental Items of Interest Pub. Co. (264 pp.). *Our copy by courtesy of the publishers.*

This book is a rather disorderly compilation of opinions both scientific and unscientific, presented to support the author's contention that the mastication of fibrous foods is the one method of preventing dental caries and malocclusion.

The main criticisms of this book are that the newer knowledge is overlooked, no consideration being given to any other factor involved in dental caries than mastication and the lack of unity and logic in the development of the arguments.

Sim Wallace is particularly concerned with the lack of significance of vitamins and heredity in the growth and development of sound oral tissues. "Oral Mal-hygiene" is the sole cause. "The administration of cod liver oil or other vitamin concentrates is quite unnecessary to ensure the proper development of the jaws" (page 38) and "it would seem wise to call a halt to the wholesale advocacy of cod-liver oil, vitamin D, or various forms of irradiated milk" (page 38), this latter statement being repeated on page 250 and being one of the countless repetitious passages with which the book abounds.

This book should be read only by the critical student of the subject who can evaluate such statements as "the old untenable and discarded belief that the incidence of caries depended on the quality of the tooth itself."

This publication cannot be recommended except to those who are well acquainted with the facts of nutrition and dental caries and who wish to familiarise themselves with the theory of Sim Wallace.

—*N. D. Martin.*

TEXTBOOK OF FUNCTIONAL JAW ORTHOPAEDICS, by K. Haupl, W. J. Grossman and P. Clarkson, London, 1952. Henry Kimpton. (408 pp., 309 illus.). *Our copy by courtesy of the publishers.*

The book is set out in the conventional manner of orthodontic textbooks, and includes chapters covering the usual aspects of the speciality. The two chapters dealing with the "Organ of Mastication and Development of the Human Dentition" are rather brief and do not give a full review of current knowledge on these aspects. The arrangement of the bone trabeculae, and the manner in which they modify themselves as a result of functional stresses is amplified. The tracing of the development of the dentition is rather brief and illustrated by the limited use of photographs, diagrams, skull dissections and models; no use is made of serial standard radiographs. This shortcoming is rather surpising as the methods of treatment in some cases are primary ones of functional influences and of guiding normal growth factors.

The chapter on terminology of malocclusion appears rather confused and requires careful study. Case classification is rather too complex and it is felt a simpler form would be quite adequate.

The chapters dealing with treatment are well set out and the logical plan of case management is easy to follow. The practical hints and descriptions are very helpful and allow for better understanding of this method of treatment.

Treatment methods are divided into:

(a) Purely functional — this deals with the Andresen activator in all its uses and covers appliance construction from both the chairside and laboratory aspects. It is shown that, although much of the appliance adjustments can be done on the models, final and accurate alterations must be made at the chairside if the best results are to be obtained from this method of treatment.
Case reports are limited and deal primarily with cases exhibiting a variation in intermaxillary relationship.

(b) Active functional—this deals with the Schwartz method of treatment and covers appliance construction and the various types of movements obtainable.

The chapter on extractions, although brief, appears to lack scientific background and it would seem to depend on the operator's practical clinical experience.

This is a well laid out textbook with an abundance of diagrams and photographs. It is probably the best book available in Australia on the functional approach to orthodontic treatment and should find a place in any practitioner's library who is interested in this branch of dentistry.

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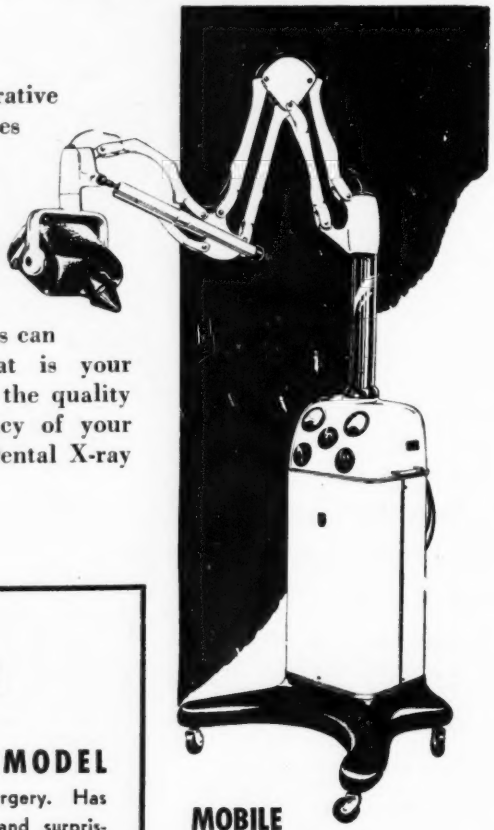

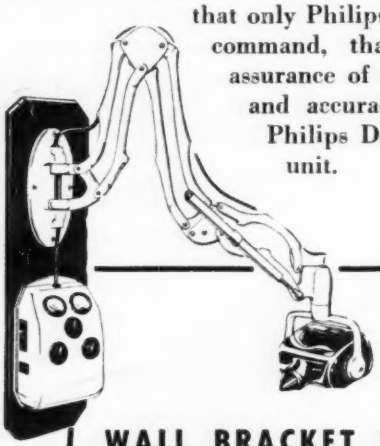

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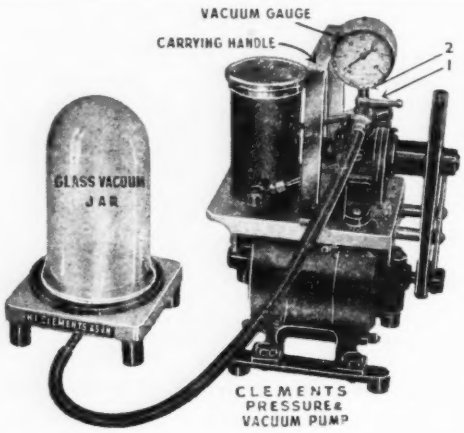

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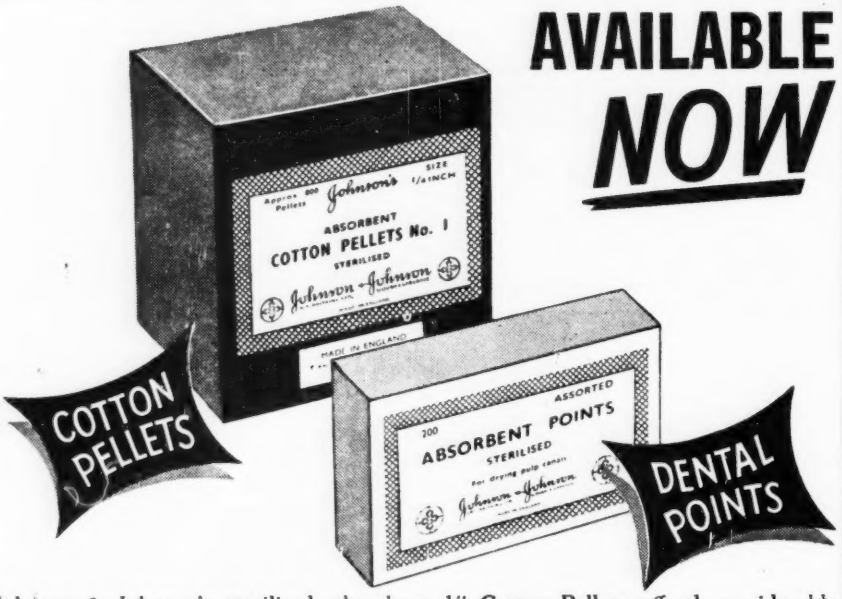

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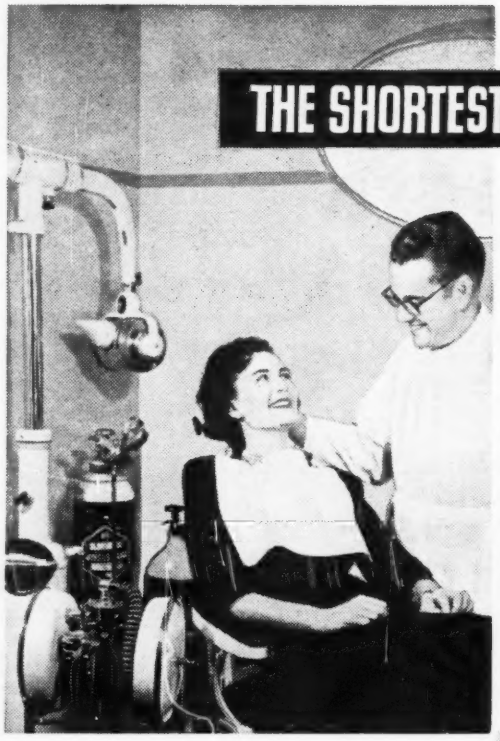

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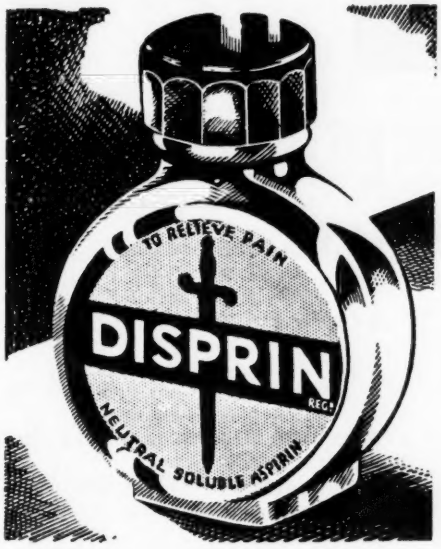

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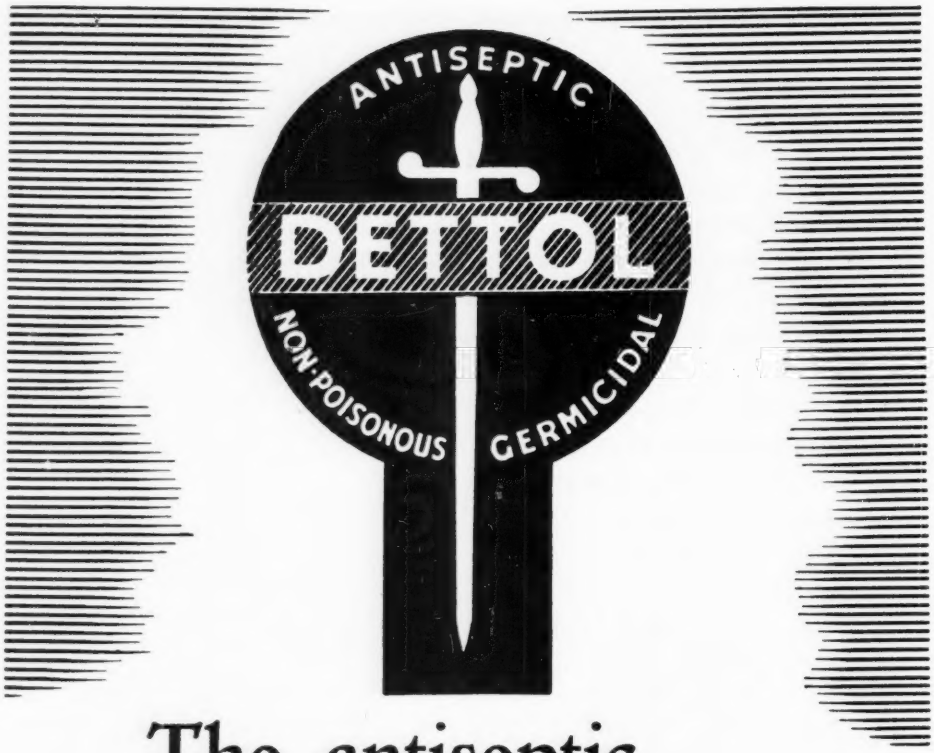

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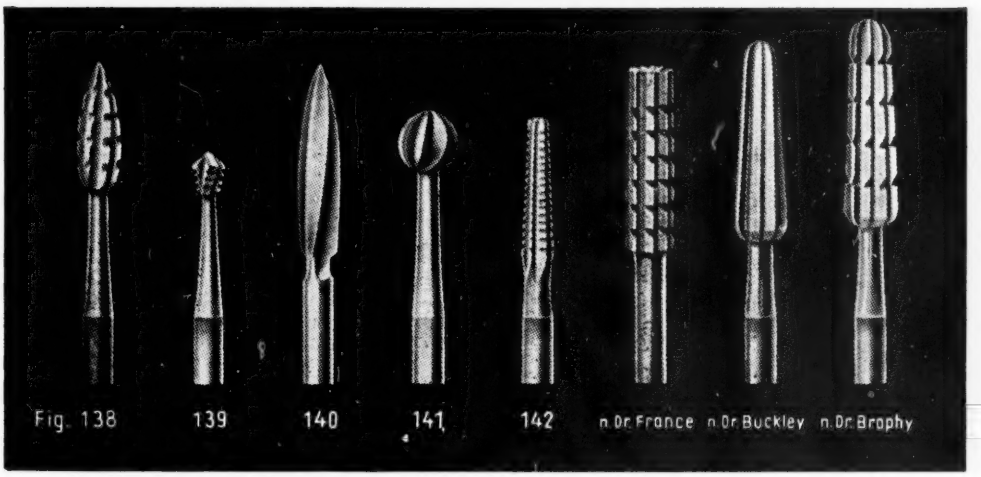

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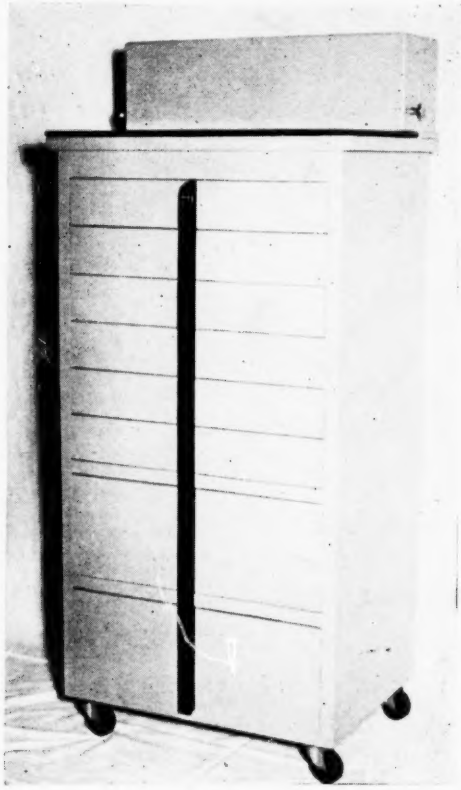

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